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L1          122 SEA FILE=REGISTRY ABB=ON  PLU=ON  LEPTIN(5A)RECEPTOR?
L2          163 SEA FILE=REGISTRY ABB=ON  PLU=ON  (OB OR OBES OR WSX OR H
                EMATOPOIET? OR HAEMATOPOIET? OR HEMOPOIET? OR HAEMOPOIET?
                )(5A)RECEPTOR?
L3          167 SEA FILE=HCAPLUS ABB=ON  PLU=ON  LEPTIN(5A)RECEPTOR? OR L
                1
L4         1559 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (OB OR OBES OR WSX OR HE
                MATOPOIET? OR HAEMATOPOIET? OR HEMOPOIET? OR HAEMOPOIET?)
                (5A)RECEPTOR? OR L2
L15           38 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L3 OR L4)(5A)(ANTIBOD?
                OR ABS OR AB OR MAB# OR PAB# OR MONOCLONAL)

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L15  ANSWER 1 OF 38  HCAPLUS  COPYRIGHT 1997 ACS
AN   1997:547405  HCAPLUS
TI   The cytokine receptor WSX, agonist and antagonist ligands and their
     uses
SO   PCT Int. Appl., 219 pp.
     CODEN: PIXXD2
IN   Bennett, Brian; Carter, Paul J.; Chiang, Nancy Y.; Kim, Kyung Jin;
     Matthews, William; Rodrigues, Maria L.
PI   WO 9725425 A1  970717
AI   WO 97-US325  970107
PY   1997
AB   The cytokine receptor WSX that plays a role in hematopoiesis is
     identified and antibodies to it (including agonist and neutralizing
     antibodies) are disclosed and uses for them are described.  Uses for
     WSX ligands (e.g., anti-WSX receptor
     agonist antibodies or OB protein) in
     hematopoiesis are also disclosed.  The gene for the receptor was
     cloned using probes derived from a human liver expressed sequence
     tag to screen a Hep3B cDNA library and a full-length clone
     constructed from several overlapping clones.  The receptor may play
     a role in control of cellular proliferation and it is expressed in
     fetus (lung, liver, kidney) and in adult (liver, placenta, lung,
     skeletal muscle, kidney, ovary, prostate, small intestine).  A no.
     of variants of the receptor were found, of which one (13.2) was a
     receptor for OB protein (leptin).  OB protein was found to interact
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synergistically with interleukin 3, stem cell factor, and GM-CSF in hematopoiesis with a preferential stimulation of myelopoiesis. The identification of agonist antibodies is described.

L15 ANSWER 2 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:499251 HCAPLUS
TI Cloning of cDNA for db gene encoding the receptor for leptin and use of the receptor
SO PCT Int. Appl., 171 pp.
CODEN: PIXXD2
IN Friedman, Jeffrey M.; Lee, Gwo-hwa; Proenca, Ricardo; Ioffe, Ella
PI WO 9726335 A1 970724
AI WO 97-US1010 970116
PY 1997
AB Disclosed is the cDNA of murine db gene encoding the leptin receptor (OB-R) that is involved in body wt. homeostasis. Mutations in this receptor that has sequence similarity to gp130 cytokine receptors are assocd. with obese phenotypes. Also described are the identification and characterization of OB-R for leptin, including a naturally occurring sol. form of OB-R that is expected to modulate leptin activity, in particular to agonize leptin activity. Use of OB-R for identification of leptin analogs, diagnosis and therapy for obese, etc., is also disclosed. The cDNA for splice variants OB-Ra, OB-Rb, OB-Rc, OB-Rd, OB-Re, and their sol. forms; and the deduced amino acid sequences are also disclosed. Substitution mutation of murine leptin receptors may be induced to prep. variants. Also disclosed are methods for recombinant prepn. of OB-R, use of ribozymes and antisense nucleic acids of OB-R, detection/detn. of OB-R, diagnosis/treatment of the diseases involved with OB-R, and body wt. control.

L15 ANSWER 3 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:457155 HCAPLUS
DN 127:90511
TI Mouse and human Ob receptors, DNA sequences, and diagnosis and treatment of body weight disorders
SO PCT Int. Appl., 260 pp.
CODEN: PIXXD2
IN Tartaglia, Louis A.; Tepper, Robert I.; Culpepper, Janice A.; et al.
PI WO 9719952 A1 970605
AI WO 96-US19128 961127
PY 1997
AB The present invention relates to the discovery, identification and characterization of nucleotides that encode Ob receptor (ObR), a receptor protein that participates in mammalian body weigh regulation. The invention encompasses obR nucleotides, host cell expression systems, ObR proteins, fusion proteins, polypeptides and peptides, antibodies to the receptor, transgenic animals that express an obR transgene, or recombinant knock-out animals that do not express the ObR, antagonists and agonists of the receptor, and other compds. that modulate obR gene expression or ObR activity that can be used for diagnosis, drug screening, clin. trial monitoring, and/or the treatment of body wt. disorders, including but not limited to obesity, cachexia and anorexia. Examples include mouse and human Ob receptors and nucleic acid sequences encoding them. Also, IgG1 fusion protein is recombinantly expressed. The mouse gene is mapped to mouse chromosome 4 and identified as the same as gene db.

L15 ANSWER 4 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:324406 HCAPLUS
DN 126:292452
TI Human hematopoietin receptor NR2 cDNA sequence, receptor ligands, recombinant production, and oligonucleotide or antibody probes
SO PCT Int. Appl., 96 pp.
CODEN: PIXXD2

IN Hilton, Douglas James; Willson, Tracy; Nicola, Nicos A.; Gainsford, Timothy; Alexander, Warren S.; Metcalf, Donald; Ng, Ashley
PI WO 9712037 A1 970403
AI WO 96-AU607 960926
PY 1997
AB The present invention is directed to a novel hematopoietin receptor or a deriv. thereof and to genetic sequences encoding same. The human hematopoietin receptor NR2 and its derivs. and the genetic sequences encoding it of the present invention are useful in the development of a wide range of agonists, antagonists, therapeutics and diagnostic reagents based on ligand interaction with its receptor. The present invention particularly relates to a receptor for leptin.

L15 ANSWER 5 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:218875 HCAPLUS
DN 126:302021
TI The leptin receptor activates Janus kinase 2 and signals for proliferation in a factor-dependent cell line
SO Mol. Endocrinol. (1997), 11(4), 393-399
CODEN: MOENEN; ISSN: 0888-8809
AU Ghilardi, Nico; Skoda, Radek C.
PY 1997
AB The **antibody** effects of **leptin** are mediated by the obese **receptor** (OB-R), a member of the cytokine receptor superfamily. Several isoforms of OB-R that differ in the length of the cytoplasmic domain have been described. An isoform with a long cytoplasmic domain of 302 amino acids, termed OB-Rb, contains the conserved box 1 and box 2 and is likely to be responsible for leptin-induced signaling. A point mutation in the OB-R gene of diabetes (db) mice generates a new splice donor that interferes with the correct splicing of the OB-Rb mRNA and is predicted to cause absence of the OB-Rb protein in db/db mice. Here the authors examd. the signaling potential of the long isoform, OB-Rb, and of a short isoform, OB-Ra, in BaF3 cells, a factor-dependent hematopoietic cell line. The long isoform was able to generate a proliferative signal upon leptin binding, activated Janus kinase 2 (Jak2). Consistently, antibodies directed against the extracellular domain of OB-R copptd. Jak2. The short isoform, OB-Ra was inactive in both proliferation and Jak activation. These results provide further support for the long isoform, OB-Rb, being the principal mediator of the effects of leptin and help to explain why db/db mice are resistant to leptin, despite the presence of the short OB-R isoforms.

L15 ANSWER 6 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:111200 HCAPLUS
DN 126:115415
TI Methods for obtaining compositions enriched for hematopoietic stem cells and antibodies for use therein
SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
IN Hill, Beth Louise; Rozler, Elen; Chen, Benjamin P.
PI WO 9640874 A1 961219
AI WO 96-EP2462 960606
PY 1996
AB A method for obtaining human hematopoietic stem cells is provided by enrichment for stem cells using a novel stem cell marker, EM5, which is expressed on stem and progenitor cells while being less accessible or absent on more mature cells. Compns. obtained thereby and reagents for use therein are also provided.

L15 ANSWER 7 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:111195 HCAPLUS
DN 126:115414
TI Methods for obtaining compositions enriched for hematopoietic stem

cells and antibodies for use therein
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
IN Hill, Beth Louise; Rozler, Elen; Chen, Benjamin P.
PI WO 9640875 A1 961219
AI WO 96-EP2463 960606
PY 1996
AB A method for obtaining human hematopoietic stem cells is provided by enrichment for stem cells using a novel stem cell marker, EM10, which is expressed on stem and progenitor cells while being less accessible or absent on more mature cells. Compns. obtained thereby and reagents for use therein are also provided.

L15 ANSWER 8 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:47656 HCAPLUS
DN 126:102928
TI Reactivity of Cytokine **Receptor** Panel **mAb** with immature **hemopoietic** cells and cell lines
SO Leucocyte Typing V: White Cell Differ. Antigens, Proc. Int. Workshop Conf., 5th (1995), Meeting Date 1993, Volume 2, 1945-1949. Editor(s): Schlossman, Stuart F. Publisher: Oxford University Press, Oxford, UK.
CODEN: 63WDAC
AU Buhring, Hans-Jorg; Burkhardt, Michaela; Ning, Yu; Zhu, Xixia; Volkmann, Rudiger; Muller, Claudia A.; Pawelec, Graham; Bruserud, Oystein
PY 1995
AB The cellular reactivities of monoclonal antibodies of the Cytokine Receptor Section were analyzed on the surface of the KG1, KG1a, UT-7, TF-1, MO7e, and MEG-01 cell lines and on CD34+ bone marrow cells, using flow cytometry. Emphasis was given to the further characterization of subpopulations of purified steel factor receptor-pos. cells from bone marrow, cord blood, and peripheral blood. Since several cytokine receptors are expressed at a detectable level only on a small subpopulation of heterogeneous cell samples, the strategy described in this report might be useful for studies with other cytokine receptor antibodies.

L15 ANSWER 9 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:6116 HCAPLUS
DN 126:27676
TI Nucleic acid constructs encoding the mouse ob polypeptide for treating obesity
SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
IN Giese, Klaus W.; Williams, Lewis T.
PI WO 9635787 A1 961114
AI WO 96-US6609 960508
PY 1996
AB A nucleic acid mol. is provided that can be expressed in a host cell to produce a biol. active ob polypeptide than can effectively inhibit food intake and/or wt. gain. Vectors and host cells contg. the nucleic acid mol. and also provided, as well as methods for producing the ob protein and other ob polypeptides, methods of induction of the prodn. of the ob polypeptides, such as by in in vivo or ex vivo gene therapy, and methods for inhibition of food intake and/or wt. gain. Thus, murine obese (ob) polypeptide cDNA contg. a 167-amino-acid open reading frame (including a signal peptide moiety) was cloned by RT-PCR and used as a template for generation of various ob constructs for protein expression in prokaryotes and eukaryotes. The ob expression constructs encoded either full-length ob protein or truncated versions lacking variously 1-20, 1-21, or 1-24 of the N-terminal amino acid residues of the ob protein. In addn., in some of the constructs, the ob coding region was fused at the C-terminus to addnl. nucleotide sequences comprising epitopes such as Myc or HA (influenza virus

hemagglutinin) for recognition with anti-Myc or anti-HA antibodies or for labeling with heart muscle kinase in the presence of [.gamma.-32P]ATP. Further provided are antibodies to the ob polypeptides and methods of using such antibodies, such as for identification, detection, or isolation of an receptor is provided as well as methods for prodn. of **antibodies** to the **ob receptor**. The antibodies and polypeptides herein can be incorporated in kits for immunoassays. Pharmaceutical compns. contg. the ob polypeptide and **antibodies** to the **ob** polypeptide or to the ob **receptor** can be used for administration to animals and humans. I.v. administration of the bacterially expressed and purified ob polypeptide of construct #1127 to CD rats resulted in no wt. gain, redn. in the animals' food intake and fecal output; slightly depressed water intake, and normal urinary output; control rats gained wt., and had higher food intake and fecal wts. than the ob-treated rats.

L15 ANSWER 10 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1997:2495 HCAPLUS
DN 126:30350
TI Human antibodies derived from immunized xenomice
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
IN Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue; Brenner, Daniel G.; Capon, Daniel J.
PI WO 9633735 A1 961031
AI WO 96-US5928 960429
PY 1996
AB Fully human antibodies against a specific antigen can be prepd. by administering the antigen to a transgenic animal which has been modified to produce such antibodies in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either antibodies per se or analogs thereof. Antibodies or monoclonal antibodies to human interleukin 6, tumor necrosis factor .alpha., CD4, L-selectin, gp39, tetanus toxin, PTH-related protein, and interleukin 8 were prepd. in xenomice.

L15 ANSWER 11 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:756546 HCAPLUS
DN 126:17804
TI Human antibodies derived from immunized xenomice
SO PCT Int. Appl., 64 pp.
CODEN: PIXXD2
IN Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue; Brenner, Daniel G.; Capon, Daniel J.
PI WO 9634096 A1 961031
AI WO 95-US5500 950428
PY 1996
AB Antibodies with fully human variable regions against a specific antigen can be prepd. by administering the antigen to a transgenic animal which has been modified to produce such antibodies in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either antibodies per se or analogs thereof.

L15 ANSWER 12 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:604878 HCAPLUS
DN 125:323779
TI Leptin. A novel fat cell-derived blood-borne satiety factor in the regulation of energy homeostasis
SO Jikken Igaku (1996), 14(16), 2236-2242
CODEN: JIIGEF; ISSN: 0288-5514
AU Ogawa, Yoshihiro; Hosoda, Kiminori; Nakao, Kazuwa
PY 1996
AB A review with 46 refs., on the mol. mechanism of leptin-mediated

regulatory system of energy homeostasis, discussing ob gene expression and leptin in adipocytes, regulation of energy homeostasis by leptin, expression and the structure of **ab** gene-encoded **leptin receptors**.

L15 ANSWER 13 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:579543 HCAPLUS
DN 125:266507
TI Evidence of free and bound leptin in human circulation. Studies in lean and obese subjects and during short-term fasting
SO J. Clin. Invest. (1996), 98(6), 1277-1282
CODEN: JCINAO; ISSN: 0021-9738
AU Sinha, Madhur K.; Opentanova, Irina; Ohannesian, Joanna P.; Kolaczynski, Jerzy W.; Heiman, Mark L.; Hale, John; Becker, Gerald W.; Bowsher, Ronald R.; Stephens, Thomas W.; Caro, Jose F.
PY 1996
AB Little is known about leptin's interaction with other circulating proteins which could be important for its biol. effects. Sephadex G-100 gel filtration elution profiles of 125I-leptin-serum complex demonstrated 125I-leptin eluting in significant proportion assocd. with macromols. The 125I-leptin binding to circulating macromols. was specific, reversible, and displaceable with unlabeled leptin (ED50:0.73 nM). Several putative leptin binding proteins were detected by leptin-affinity chromatog. of which either 80- or 100-kDa proteins could be the sol. leptin receptor as .apprx.10% of the bound 125I-**leptin** was immunoprecipitable with **leptin receptor antibodies**. Significantly higher proportions of total leptin circulate in the bound form in lean (46.5%) compared with obese (21.4%) subjects. In lean subjects with 21% or less body fat, 60-98% of the total leptin was in the bound form. Short-term fasting significantly decreased basal leptin levels in three lean and three obese subjects while refeeding restored it to basal levels. The effects of fasting on free leptin levels were more pronounced in lean subjects (basal vs. 24-h fasting: 19.6 vs. 1.3 ng/mL) compared with those in obese subjects (28.3 vs. 14.7). No significant decrease was obsd. in bound leptin in either group. These studies suggest that in obese individuals the majority of leptin circulates in free form, presumably bioactive protein, and thus obese subjects are resistant to free leptin. In lean subjects with relatively low adipose tissue, the majority of circulating leptin is in the bound form and thus may not be available to brain receptors for its inhibitory effects on food intake both under normal and food deprivation states.

L15 ANSWER 14 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:354059 HCAPLUS
DN 125:26943
TI Hu-B1.219, a novel human hematopoietin receptor and a cDNA encoding it
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
IN Snodgrass, Ralph H.; Cioffi, Joseph; Zupancic, Thomas J.; Shafer, Alan W.
PI WO 9608510 A1 960321
AI WO 95-US10965 950830
PY 1996
AB A novel member of the hematopoietin receptor family, Hu-B1.219, and a cDNA encoding it are characterized. Host cells that express the Hu-B1.219 coding sequence may be used to evaluate and screen for ligands or drugs involved in Hu-B1.219 interaction and regulation. Expression has been detected in certain human fetal tissues and cancer cells, mol. probes designed from its nucleotide sequence may be useful for prenatal testing and cancer diagnosis. The gene is strongly expressed in fetal lung and liver and adult heart, lung, liver and ovary. It is also expressed in the tumor cell lines K-562

and A549. The gene and protein are similar to other members of the hemopoietin receptor family but are distinct from them.

L15 ANSWER 15 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:164780 HCAPLUS
DN 124:229441
TI Immobilized anti-KIT monoclonal antibody induces ligand-independent dimerization and activation of steel factor receptor: biologic similarity with membrane-bound form of steel factor rather than its soluble form
SO Blood (1996), 87(6), 2235-43
CODEN: BLOOAW; ISSN: 0006-4971
AU Kurosawa, Kazuyo; Miyazawa, Keisuke; Gotoh, Akihiko; Katagiri, Tomoko; Nishimaki, Jiroh; Ashman, Leonie K.; Toyama, Keisuke
PY 1996
AB Interaction of a tyrosine kinase type receptor and its ligand induces receptor-dimerization or -oligomerization followed by transphosphorylation and activation of its intrinsic kinase, which leads to a series of intracellular signals. We have previously reported that the membrane-bound form of Steel factor (SLF) induces more persistent tyrosine kinase activation and longer life span of c-kit encoded protein (KIT) than its sol. form (Miyazawa et al., Blood 85:641, 1995). In this study, we used YB5.B8 monoclonal antibody (MoAb) that recognizes the extracellular domain of KIT to investigate whether immobilized anti-KIT MoAb can substitute for SLF as a potent activator of KIT by crosslinking receptors and further compared its effect with each SLF isoform in a factor-dependent cell line MO7e. YB5.B8 MoAb in a sol. state suppressed SLF-induced MO7e cell proliferation in a dose-dependent manner. By contrast, once this antibody was immobilized on the goat-antimouse MoAb (GAM)-coated culture plates, it supported the growth of MO7e cells in the absence of any growth factors, whereas culturing the cells in GAM alone or YB5.B8 without GAM-coated plates resulted in rapid cell-death within 24 h. As with the natural ligand SLF, immobilized YB5.B8 MoAb synergized with granulocyte-macrophage colony-stimulating factor (GM-CSF) in inducing cell proliferation compared with either YB5.B8 MoAb or GM-CSF alone. Immunoblotting with antiphosphotyrosine MoAb showed that interaction of MO7e cells with immobilized YB5.B8 induced tyrosine phosphorylation of a series of intracellular proteins including KIT (145 kD). In addn., crosslinking studies using a water-sol. crosslinking reagent bis-sulfosuccinimidyl-suberate showed that immobilized YB5.B8 MoAb induced dimerization and activation of KIT. However, as with stimulation by the membrane-bound form of SLF, the kinetics of KIT activation with YB5.B8 MoAb was more prolonged compared with the cells treated with recombinant sol. SLF. Flow cytometry showed that, unlike the cells treated with sol. SLF, no downmodulation of cell-surface KIT expression was obsd. in MO7e cells cultured with immobilized YB5.B8 MoAb. These data suggest that immobilized **antibodies** against **hematopoietic receptors** may replace their ligand-stimulators; however, their activities may resemble the membrane-bound form rather than the sol. form of natural ligands.

L15 ANSWER 16 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1996:4051 HCAPLUS
DN 124:84368
TI 71E1, anti-flk-2 tyrosine kinase receptor
SO Hybridoma (1995), 14(5), 513
CODEN: HYBRDY; ISSN: 0272-457X
AU Goldstein, Neil
PY 1995
AB A hybridoma was generated which produces rat IgG2b class monoclonal antibodies specific for the human flk-2 tyrosine kinase receptor. Human flk-2-transfected 3T3 cells were used as the immunizing antigen.

L15 ANSWER 17 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1995:990846 HCAPLUS
DN 124:28043
TI Agonist antibodies against the flk2/flt3 receptor and uses thereof
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
IN Bennett, Brian D.; Broz, Susan D.; Matthews, William; Zeigler, Francis C.
PI WO 9527062 A1 951012
AI WO 95-US3718 950323
PY 1995
AB Agonist antibodies are disclosed which bind to the extracellular domain of the flk2/flt3 receptor and thereby activate the intracellular kinase domain thereof. The labeled antibodies are useful as diagnostics for detecting the presence of the flk2/flt3 receptor in primitive hematopoietic cells for example. The antibodies are able to cause primitive hematopoietic cells to proliferate and/or differentiate and thereby enhance repopulation of mature blood cell lineages in a mammal which has undergone chemotherapy or radiation therapy or bone marrow transplantation. The antibodies are further useful for treating mammals which have suffered a decrease in blood cells as a consequence of disease or a hemorrhage, for example.

L15 ANSWER 18 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1995:630105 HCAPLUS
DN 123:31242
TI **Monoclonal antibodies** that recognize flk-2 **receptors** and the isolation of primitive **hematopoietic** stem cell populations
SO PCT Int. Appl., 48 pp.
CODEN: PIXXD2
IN Goldstein, Neil I.; Songsakphisan, Ratchanee; Rose, Caroline
PI WO 9507348 A1 950316
AI WO 94-US10194 940907
PY 1995
AB Antibodies that bind specifically to the flk-2 tyrosine kinase receptor may be used to isolate a population of hematopoietic stem cells that expresses the flk-2 receptor. In example, flag flk-2 plasmids were constructed and expressed in COS monkey cells, recombinant flag-flk-2 proteins were purified and used for raising anti-mouse and anti-human flk-2 monoclonal antibodies, and the antibodies were used in immunoassay. The antibodies can be used for sepg. hematopoietic stem cells that are CD34+, flk-2+, and/or Lin-Thy-1+.

L15 ANSWER 19 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1995:341047 HCAPLUS
DN 122:103938
TI Monoclonal antibody to interleukin-7 (IL-7) receptor, process for producing the same, and use thereof
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
IN Nishikawa, Satomi; Sudo, Tetsuo; Okano, Kiyoshi; Izawa, Akiko; Nakamura, Noriko; Akiyama, Naoko
PI WO 9428160 A1 941208
AI WO 94-JP887 940601
PY 1994
AB A monoclonal antibody specific for mouse IL-7 receptors is prepd. by the hybridoma method. The monoclonal antibody is useful for immunoassay of the IL-7 receptors with high sensitivity and accuracy. An artificially induced model prepd. by administering the above antibody to an animal can be utilized in elucidating the mechanism of lymphocyte differentiation and proliferation, developing an immunosuppressant, and investigating organ

transplantation, arthritis, diabetes, and so forth. Further an artificially induced model prepd. by administering the above antibody in combination with an anti-SCF receptor antibody to an animal can be utilized in elucidating diseases due to an abnormal hematopoietic function such as aplastic anemia exhibiting decreased myelopoiesis and whole blood hematopenia in the peripheral blood and in developing medicines capable of curing these blood cell disorders. A mouse contg. the above antibody in combination with an anti-SCF **receptor antibody** administered thereto has concd. **hematopoietic** stem cells, and hence the myeloma or splenic cells thereof can be used to est. hematopoietic stem cell proliferation factors. Therefore, the invention method of estn. is useful in searching for such factors.

L15 ANSWER 20 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1994:267604 HCAPLUS
DN 120:267604
TI Assessment of the anti-c-kit monoclonal antibody YB5.B8 in affinity magnetic enrichment of human lung mast cells
SO J. Immunol. Methods (1994), 169(2), 153-61
CODEN: JIMMBG; ISSN: 0022-1759
AU Okayama, Yoshimichi; Hunt, Tim C.; Kassel, Olivier; Ashman, Leonie K.; Church, Martin K.
PY 1994
AB The monoclonal antibody, YB5.B8 binds to the second domain of the c-kit proto-oncogene product on human mast cells, a receptor assocd. with tyrosine kinase activity. This mol. is involved with cell proliferation, maturation and viability as well as cell activation and its natural ligand is stem cell factor (SCF). The authors have used this antibody coupled to Dynabeads to perform pos. affinity enrichment of human lung mast cells. This procedure results in enrichment of mast cells from 2.6% to 85.0% purity with yields of 41.9%. As YB5.B8 interacts with the same receptor domain as does SCF, it is important to demonstrate that this procedure does not modify mast cell function. Incubation of mast cells with 1-5000 ng/mL YB5.B8 for 30 min neither induced histamine release nor modulated histamine release induced by anti-IgE. Furthermore, incubation with YB5.B8 did not alter prolonged culture with SCF. Examn. of cells enriched using YB5.B8 showed that they had a normal histamine content (3.8 pg/cell compared with 3.9 pg/cell unpurified) and had unchanged behavior in both histamine secretion and cell survival studies. These studies indicate that YB5.B8 does not influence mast cell function and thus its use in magnetic affinity purifn. procedures offers a simple and effective method for enriching human mast cell prepns.
IT **138359-29-2**, c-Kit receptor tyrosine kinase
RL: USES (Uses)
(**monoclonal** antibody YB5.B8 to, Dynabead conjugates of, magnetic affinity purifn. of human lung mast cells by)

L15 ANSWER 21 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1994:214877 HCAPLUS
DN 120:214877
TI Epitope mapping and functional studies with three monoclonal antibodies to the C-KIT receptor tyrosine kinase, YB5.B8, 17F11, and SR-1
SO J. Cell. Physiol. (1994), 158(3), 545-54
CODEN: JCLLAX; ISSN: 0021-9541
AU Ashman, Leonie K.; Buehring, Hans Joerg; Aylett, Gabriella W.; Broudy, Virginia C.; Muller, Claudia
PY 1994
AB Three monoclonal antibodies (MAbs) to the human c-kit receptor tyrosine kinase (P145c-kit), derived in independent labs., have been extensively used in studies of c-kit expression and the role of its ligand, steel factor (SLF), in hemopoiesis and mast cell differentiation and function. In this study, the relationship

between the epitopes they identify, and their effects on SLF binding, receptor internalization, and signal transduction are compared. Epitope mapping studies carried out on the high P145c-kit-expressing cell line HEL-DR showed that SR-1 identifies an epitope independent of those bound by YB5.B8 and 17F11, while the latter two antibodies bound to distinct but interacting epitopes. SR-1 potently blocked the binding of SLF to P145c-kit on these cells and also on cells of the factor-dependent line MO7e. In contrast, YB5.B8 and 17F11 had minimal effects on ligand binding. Conversely, SLF partially blocked the binding of SR-1 and YB5.B8 to cells, while binding of 17F11 was actually enhanced by SLF on some target cells. Preincubation of HEL-DR and MO7e cells with MAbs prior to exposure to SLF revealed that 17F11 itself brought about partial down-regulation of P145c-kit and did not inhibit SLF-mediated down-regulation. SR-1 caused minimal down-regulation and inhibited SLF-mediated receptor internalization. YB5.B8 had minimal effects on either cell line in this assay. To det. whether the antibodies had any agonist activity, they were compared with SLF for their ability to bring about receptor phosphorylation in intact MO7e cells. All three antibodies induced detectable tyrosine phosphorylation with 17F11 being the most effective, while YB5.B8 was the least effective. Finally, the ability of the antibodies to influence the proliferation of the MO7e cells was examd. As expected, SR-1 potently inhibited the proliferative response to SLF, while 17F11 weakly inhibited and YB5.B8 had negligible effect. In the absence of SLF both 17F11 and YB5.BB displayed very weak but reproducible agonist activity.

L15 ANSWER 22 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1993:79358 HCAPLUS
DN 118:79358
TI Monoclonal antibodies against c-kit
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
IN Bartke, Ilse; Kostka, Guenter; Naujoks, Kurt; Ullrich, Axel
PI WO 9221766 A1 921210
AI WO 92-EP1117 920520
PY 1992
AB Monoclonal antibodies are provided which bind to the human c-kit receptor glycoprotein which binds hematopoietic cell growth factor KL [mast cell growth factor, stem cell factor (SCF)], and inhibit binding of the growth factor by the receptor and the assocd. activation of receptor tyrosine kinase, which has an important role in neoplastic transformation. The antibodies are useful diagnostically in detection of changes in c-kit receptor expression and thereby in detg. the malignancy of hematopoietic cell tumors, seminomas, and small-cell lung carcinoma. The antibodies were produced by the std. hybridoma method from spleen cells of mice immunized with NIH-3T3 cells transfected with the human c-kit gene. They inhibited the binding of human SCF by lymphocyte c-kit receptors and the induction of lymphocyte proliferation by SCF.

L15 ANSWER 23 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:649900 HCAPLUS
DN 117:249900
TI Monoclonal antibodies to stem cell factor (SCF) receptors
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
IN Lin, Nancy; Broudy, Virginia C.
PI WO 9217505 A1 921015
AI WO 92-US2674 920403
PY 1992
AB A monoclonal antibody (SR-1) to the human SCF receptor (identified as proto-oncogene c-kit) of hematopoietic precursor cells binds to the receptor and inhibits binding of SCF to the receptor. Hematopoietic cells are sepd. from other cells, for use in bone

marrow transplantation, by utilizing their affinity for the above antibody in column chromatog., fluorescence-activated cell sorting, or immune adherence methods. Leukemia and solid tumors are treated by administration of SR-1 or a binding fragment thereof conjugated to an appropriate antineoplastic agent. Thus, mice were immunized with OCIM1 human erythroleukemia cells bearing SCF receptors for prodn. of spleen-myeloma hybrid cells by fusion; genomic DNA from the hybridomas was used to produce chimeric monoclonal antibodies having murine variable regions and human const. regions by recombinant DNA techniques.

L15 ANSWER 24 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:610395 HCAPLUS
DN 117:210395
TI Heterogeneity of immunoglobulin-associated molecules on human B cells identified by monoclonal antibodies
SO Proc. Natl. Acad. Sci. U. S. A. (1992), 89(18), 8522-6
CODEN: PNASA6; ISSN: 0027-8424
AU Nakamura, Tetsuya; Kubagawa, Hiromi; Cooper, Max D.
PY 1992
AB Two covalently linked transmembrane mols., encoded in mice by the mb-1 and B29 genes, have been defined as integral components of the antibody receptor units expressed on B cells. Monoclonal antibodies were produced against an exposed extracellular epitope on the putative human equiv. of the mouse B29 product. These antibodies, CB3-1 and -2, were used to show that cytoplasmic expression of this mol. begins in human pro-B cells (terminal deoxynucleotidyltransferase-pos., .mu. chain-neg.), whereas surface expression coincides strictly with surface immunoglobulin expression of all isotypes. Immunochem. anal. of the human Ig-assocd. mols. revealed greater mol. heterogeneity than has been noted for the murine analogs. This mol. heterogeneity of Ig-assocd. mols. varied as a function of differentiation stage and the Ig isotypes expressed by B-lineage cells. The data support the hypothesis that biochem. heterogeneity of the surface Ig-assocd. mols. may contribute to the variability in biol. effects of antigen receptor crosslinkage on B cells of different maturational stages. Because the CB3 antibodies are capable of down-modulating the antigen receptors on all B cells, they may prove therapeutically useful as universal B-cell suppressants.

L15 ANSWER 25 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:564833 HCAPLUS
DN 117:164833
TI A hematopoietic growth factor receptor-related protein from the envelope of a virus causing myeloproliferative disease in the mouse
SO PCT Int. Appl., 77 pp.
CODEN: PIXXD2
IN Charon, Martine; Gisselbrecht, Sylvie; Penciolelli, Jean Francois; Souyri, Michele; Tambourin, Pierre; Varlet, Paule; Vigon, Isabelle; Wendling, Francoise
PI WO 9207074 A1 920430
AI WO 90-FR762 901019
PY 1992
AB A protein that acts as a growth factor receptor and can become involved in differentiation and proliferation of hematopoietic cell lines is identified in the envelope of MPLV virus and a gene encoding it is cloned and expressed in animal cell culture.

L15 ANSWER 26 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:542983 HCAPLUS
DN 117:142983
TI Inhibition of hematopoietic tumor growth by combined treatment with deferoxamine and an IgG monoclonal antibody against the transferrin receptor: evidence for a threshold model of iron deprivation toxicity

SO Cancer Res. (1992), 52(15), 4144-8
CODEN: CNREA8; ISSN: 0008-5472
AU Kemp, John D.; Thorson, John A.; Stewart, Barbara C.; Naumann, Paul W.
PY 1992
AB Recent studies have suggested that iron deprivation may represent a useful new approach in cancer therapy, and several strategies for producing such deprivation are now under investigation. The authors recently provided evidence that combined treatment with the iron chelator deferoxamine and an IgG **monoclonal antibody** against the transferrin **receptor** (ATRA) produces synergistic inhibition of **hematopoietic** tumor cell growth in vitro (J. D. Kemp, K. M. Smith, L. J. Kanner et al., 1900). The current study is an attempt to analyze the mechanisms responsible for the synergistic interaction. The data show that a single IgG ATRA can produce up to 75% inhibition of iron uptake while having little effect on DNA synthesis; this suggests that tumor cells either take up or have stored amts. of iron well in excess of that required to support immediate metabolic needs. When deferoxamine and the IgG ATRA are used together, the effects on iron acquisition and receptor down-modulation are either additive or subadditive but are clearly not synergistic. Overall, the findings suggest that the IgG ATRA produces an injury to iron uptake that is just below a crit. threshold and that the addnl. effect provided by the iron chelator is sufficient to exceed that threshold and produce a rapid depletion of iron pools that are vital for short-term DAN synthesis. IgG ATRAS thus seem to be of even greater interest as therapeutic reagents, and further study of their properties and of how they interact with deferoxamine appears to be warranted.

L15 ANSWER 27 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:104026 HCAPLUS
DN 116:104026
TI Isolation and characterization of a monoclonal antibody that recognizes the human c-kit receptor
SO Blood (1992), 79(2), 338-46
CODEN: BLOOAW; ISSN: 0006-4971
AU Broudy, Virginia C.; Lin, Nancy; Zsebo, Krisztina M.; Birkett, Neal C.; Smith, Kent A.; Bernstein, Irwin D.; Papayannopoulou, Thalia
PY 1992
AB Stem cell factor (SCF) stimulates the growth of burst-forming unit-erythroid (BFU-E) and colony-forming unit granulocyte-macrophage (CFU-GM) by binding to a specific cell surface receptor. The receptor for SCF is encoded by the protooncogene c-kit. After immunizing mice with the human erythroleukemia cell line OCIM1, a monoclonal antibody (MoAb) was obtained that recognizes the human c-kit receptor. This MoAb, designated SR-1, blocks binding of 125I-human SCF to the c-kit receptor, and neutralizes the biol. effects of SCF in hematopoietic colony assays. With few exceptions, c-kit expression was identified on all hematopoietic and lymphoid cell lines tested by indirect immunofluorescent anal. using SR-1 and by binding studies with 125I-SCF. SR-1 recognizes a small fraction of normal bone marrow mononuclear cells, and these cells have the morphol. appearance of blasts. Colony assays show that BFU-E and CFU-GM display the c-kit receptor. SR-1 does not cross-react with murine c-kit protein, indicating that the binding epitopes of the human and murine c-kit receptors are antigenically distinct. This MoAb may be useful to characterize the spectrum of cells that display the c-kit receptor and to further define the role of SCF in hematopoiesis.

L15 ANSWER 28 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:94777 HCAPLUS
DN 114:94777
TI Cytotoxic activity of an anti-transferrin receptor immunotoxin on normal and leukemic human hematopoietic progenitors

SO Cancer Res. (1991), 51(2), 536-41
CODEN: CNREA8; ISSN: 0008-5472
AU Cazzola, Mario; Bergamaschi, Gaetano; Dezza, Laura; D'Uva, Roberto; Ponchio, Luisa; Rosti, Vittorio; Ascari, Edoardo
PY 1991
AB The process of cellular iron uptake involves a specific receptor for the plasma carrier transferrin and a pathway of receptor-mediated endocytosis. Transferrin receptor expression is closely related to the rate of cell proliferation, and conjugates between anti-transferrin receptor monoclonal antibodies and toxins have been shown to have potent cytotoxic activity. An anti-transferrin receptor immunotoxin has been prepd. by conjugating the anti-transferrin receptor monoclonal antibody B3/25 to a ribosome-inactivating protein, the saporin-6 (SO6), which is derived from the seeds of the plant Saponaria officinalis. The immunotoxin B3/25-SO6 was tested for in vitro cytotoxic activity against the human cell lines K-562 and HL-60 and against normal human bone marrow hematopoietic progenitors and acute myeloid leukemia clonogenic cells. The immunotoxin proved to be an effective inhibitor of K-562 and HL-60 clonogenic cell growth, in vitro colony formation being completely inhibited at immunotoxin concns. ranging from 10-7 to 10-1- M. B3/25-SO6 markedly reduced the recloning efficiency of HL-60 clonogenic cells at 10-12 M. Exposure of HL-60 cells in suspension culture to 10-9 M B3/25-SO6 for 48-72 h completely abolished their clonogenic potential. The immunotoxin was also found to be cytotoxic against normal human bone marrow progenitor cells (burst-forming unit-erythroid and colony-forming unit-granulocyte, macrophage) in concn.-dependent manner. However, exposure of normal colony-forming unit-granulocyte, macrophage in suspension culture to 10-9 M B3/25-SO6 for 72 h resulted in only 50% suppression of their clonogenic potential. Finally, B3/25-SO6 was found to be a potent inhibitor of in vitro growth of acute myeloid leukemia clonogenic cells. The cytotoxic effects of B3/25-SO6 were shown to be specific, since both saporin alone and irrelevant immunotoxins did not have any effect in the cellular systems examd. Apparently, the immunotoxin B3/25-SO6 has dose-related cytotoxic effects on both normal and leukemic human hematopoietic progenitors. Since there are substantial differences between normal and leukemic progenitors with respect to the proportion of cycling cells and the expression of transferrin receptors, B3/25-SO6 or similar immunotoxins may have clin. application in bone marrow-purging procedures.

L15 ANSWER 29 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:94719 HCAPLUS
DN 114:94719
TI Inhibition of hematopoietic progenitor colony growth by a monoclonal antibody against the transferrin receptor: comparison of unconjugated antibody with an immunotoxin containing recombinant ricin A chain
SO Int. J. Cell Cloning (1990), 8(5), 368-76
CODEN: IJCCE3; ISSN: 0737-1454
AU Shannon, Kevin M.; Ring, David B.; Houston, L. L.; Schaffner, Vann; Naylor, Juliette; Torkildson, Joseph C.; Reid, Shirley Ann; Larrick, James
PY 1990
AB The ability of an immunotoxin consisting of recombinant ricin A chain (rRA) conjugated to 454A12 MoAb, a monoclonal antibody which recognizes an epitope on the human transferrin receptor (454A12 MoAb-rRA) to inhibit the growth of erythroid burst-forming units (BFU-e) and myeloid colony-forming units (CFU-c) was compared with that of unconjugated 454A12 MoAb. A redn. in BFU-e colony growth was obsd. at 0.001 .mu.g/mL of 454A12 MoAb-rRA vs. 0.1 .mu.g/mL of unconjugated 454A12 MoAb. Comparison of the effects of 454A12 MoAb-rRA and 454A12 MOAb on myeloid colony development gave markedly different results. Unconjugated antibody had no effect on CFU-c

colony growth; in contrast, 0.01 .mu.g/mL of 454A12 MoAb-rRA reduced the no. of colonies from 139 per 1 .times. 105 to 75 per 1 .times. 105 cells plated. No myeloid progenitor colonies developed at 0.1 .mu.g/mL of immunotoxin. These observations suggest that 454A12 MoAb-rRA inhibits growth by a potent, ricin A chain-mediated toxic effect on any proliferating cells expressing transferrin receptors, whereas the 454A12 MoAb exerts a selective inhibitory effect primarily on erythroid progenitors by perturbing the transferrin cycle. While growth factor receptors expressed on hematopoietic cells represent promising targets for immunotoxin therapy, these data indicate that an immunotoxin could inhibit cellular proliferation by a different mechanism than the corresponding unconjugated MoAb. Depending on the antibody used, these differences may be important in trials using immunotoxins for in vivo treatment or in vitro purging of malignant hematopoietic cells.

L15 ANSWER 30 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:79524 HCAPLUS
DN 114:79524
TI Megaloblastic hematopoiesis in vitro. Interaction of anti-folate **receptor antibodies** with **hematopoietic** progenitor cells leads to a proliferative response independent of megaloblastic changes
SO J. Clin. Invest. (1991), 87(1), 313-25
CODEN: JCINAO; ISSN: 0021-9738
AU Antony, Asok C.; Briddell, Robert A.; Brandt, John E.; Straneva, John E.; Verma, Rama S.; Miller, Michael E.; Kalasinski, Lorrie A.; Hoffman, Ronald
PY 1991
AB This study tested the hypothesis that anti-placental folate receptor (PFR) antiserum-mediated effects on hematopoietic progenitor cells in vitro of increased cell proliferation and megaloblastic morphol. were independent responses. The authors detd. that (a) purified IgG from anti-PFR antiserum reacted with purified apo- and holo-PFR and specifically immunopptd. a single (44-kD) iodinated moiety on cell surfaces on low-d. mononuclear cells (LDMNC); (b) when retained in culture during in vitro hematopoiesis, anti-PFR IgG (in contrast to PFR-neutralized anti-PFR IgG and nonimmune IgG) consistently led to increased cloning efficiency of colony forming unit-erythroid (CFU-E), burst forming unit-E (BFU-E), CFU-granulocyte macrophage (CFU-GM), and CFU-GEM megakaryocyte (CFU-GEMM), and objectively defined megaloblastic changes in orthochromatic normoblasts from CFU-E- and BFU-E-derived colonies; (c) when anti-PFR antiserum was removed after initial (<1 h) incubation with LDMNC, a cell proliferation response was induced, but megaloblastic changes were not evident. (D) conversely, delay at 4.degree. for 24 h before long-term plating with antiserum resulted in megaloblastosis without increased cell proliferation; (e) however, 500-fold molar excess extracellular folate concns. completely abrogated the expected anti-PFR antiserum-induced megaloblastic changes, without altering cell proliferative responses. Thus, although cell proliferative and megaloblastic changes are induced after short-term and prolonged interaction of **antibody** with folate **receptors** on **hematopoietic** progenitors, resp., they are independent effects.

L15 ANSWER 31 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:110352 HCAPLUS
DN 110:110352
TI Characterization of a novel form of transferrin receptor preferentially expressed on normal erythroid progenitors and precursors
SO Blood (1989), 73(1), 214-21
CODEN: BLOOAW; ISSN: 0006-4971
AU Cotner, T.; Das Gupta, A.; Papayannopoulou, T.; Stamatoyannopoulos, G.

PY 1989
AB A panel of monoclonal antibodies (MoAbs) against cell surface proteins of early BFUe progeny was characterized. Five of these antibodies (Abs) reacted with normal erythroid, but not myeloid, bone marrow cells. Each of the 5 antibodies, typified by Ab 69.20, immunopptd. a dimeric complex of 185,000, which is composed of 2 identical S-S bonded subunits. This antigen had affinity for transferrin, and was essentially identical in biochem. characteristics to transferrin receptors pptd. with the well-characterized MoAbs OKT9 and 5E9. However, this form of transferrin receptor lacked both the OKT9 and 5E9 antigenic determinants and, moreover, the 69.20 epitope was absent from the conventional transferrin receptor, as defined by Abs OKT9 and 5E9. Modulation expts. demonstrated that both 69.20 and OKT9 modulated large, virtually independent populations of transferrin receptors. Both forms of transferrin receptor appeared to be derived from the product of a single gene, but the form defined by MoAb 69.20 apparently predominates in cells of the erythroid lineage and some transformed cell types that manifest a special requirement for Fe. These data suggest that cells with a high Fe requirement synthesize 2 forms of transferrin receptor, possibly by means of differential mRNA splicing or by posttranslational modification of the transferrin receptor.

L15 ANSWER 32 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1986:166481 HCAPLUS
DN 104:166481
TI A radioimmunoassay that sandwiches human interleukin-2 between radiolabeled **monoclonal antibody** and the **receptor** on a **hematopoietic** cell line
SO J. Immunol. Methods (1986), 87(2), 245-9
CODEN: JIMMBG; ISSN: 0022-1759
AU Ohike, Yoshimoto; Imai, Mitsunobu; Tanaka, Eiji; Mukaida, Naofumi; Kasahara, Tadashi; Tachibana, Katsumi; Miyakawa, Yuzo; Mayumi, Makoto
PY 1986
AB Two monoclonal antibodies were raised against human interleukin-2 (IL-2) produced by Escherichia coli contg. recombinant cDNA. Neither antibody neutralized IL-2 activity, nor did they inhibit the binding of IL-2 to the receptor on target cells. Taking advantage of the ability of monoclonal antibodies to detect IL-2 that had bound to the receptor, an RIA was developed that sandwiched IL-2 between the radiolabeled **monoclonal antibody** and the **receptor** on a **hematopoietic** cell line infected with human T cell leukemia virus Type I. The assay had the advantage of detecting only IL-2 with the ability to bind to the receptor, and displayed a linear dose-response relationship over concns. ranging from 5 to 100 ng/mL.

L15 ANSWER 33 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1986:127926 HCAPLUS
DN 104:127926
TI Distinct reactivities of four monoclonal antibodies with human interleukin 2 receptor
SO Microbiol. Immunol. (1985), 29(10), 959-72
CODEN: MIIMDV; ISSN: 0385-5600
AU Tanaka, Yuetsu; Tozawa, Hideki; Hayami, Masanori; Sugamura, Kazuo; Hinuma, Yorio
PY 1985
AB Two new murine monoclonal IgG1 antibodies, H-31 and H-A26, were characterized in comparison with 2 previously obtained monoclonal antibodies against human interleukin 2 (IL-2) receptor (IL-2 R), anti-Tac and HIEI (a monoclonal antibody that recognizes Tac antigen). In immunofluorescence assays with various human hematopoietic cells, H-31 and H-A26 antibodies both reacted with only IL-2 R-pos. cells, and they pptd. IL-2 R mols., glycoproteins

with mol. wts. of 60 kilodaltons and 53 kilodaltons (gp60/gp53), from human T-cell leukemia virus type I (HTLV-I)-carrying MT-2 cells, as demonstrated by sequential immunopptn. after absorption of IL-2 R with anti-Tac. Antibody-binding competition assays showed that H-31 and anti-Tac, and H-A26 and HIEI, resp., competed reciprocally in binding to the cells, and that anti-Tac also inhibited the binding of HIEI but not vice versa. H-31, like anti-Tac, strongly inhibited the IL-2-dependent proliferation of normal activated T-cells, absorption of IL-2 and direct binding of IL-2 to the cells, whereas H-A26, like HIEI, inhibited those processes only weakly. The spectra of reactivities of these antibodies with various simian cell lines derived by HTLV-I infection were different, as revealed by immunofluorescence studies. Human IL-2 R expresses a unique antigenic determinant, detected with HIEI, that was not detectable in IL-2 R mols. of Old and New World monkeys, and also expresses determinants common to simian IL-2 R mols. Thus, antibodies H-31 and H-A26 recognize human IL-2 R mols. and the antigenic sites on the IL-2 R mol. defined by H-31, H-A26, anti-Tac, and HIEI are different.

L15 ANSWER 34 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1985:111146 HCAPLUS
DN 102:111146
TI Analysis of lymphopoietic stem cells with a monoclonal antibody to the rat transferrin receptor
SO Immunology (1985), 54(2), 333-41
CODEN: IMMUAM; ISSN: 0019-2805
AU Jefferies, Wilfred A.; Brandon, M. R.; Williams, A. F.; Hunt, S. V.
PY 1985
AB A mouse monoclonal IgG2a antibody, designated MR COX-26, is specific for the rat transferrin receptor, but does not block transferrin binding. The antibody labeled a myeloma, 3 leukemia cell lines and normal dividing cells of various types, but also bound to a no. of nondividing normal tissues. No labeling of lymphopoietic stem cells could be detected, even though approx. 25% of bone marrow and >95% of fetal liver cells were clearly labeled.

L15 ANSWER 35 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1984:136754 HCAPLUS
DN 100:136754
TI The relation of iron uptake to the proliferation of erythroid and hemopoietic cells
SO Biomed. Biochim. Acta (1983), 42(11-12, Suppl.), 177-81
CODEN: BBIADT
AU Neuwirt, J.; Hradilek, A.; Bartek, J.
PY 1983
AB Treatment of several **hematopoietic** cell lines with transferrin-**receptor**-blocking or Fe-uptake-inhibiting **monoclonal** antibodies to human transferrin suggested that both transferrin binding to receptors and transferrin-mediated Fe delivery to the cells are essential for cell growth and proliferation. A HeLa cell subline adapted for growth in serum-free low-Fe medium took up 3-5-fold more Fe from Fe citrate (1-10 .mu.M) than did the parental cells. The role of transferrin-dependent and transferrin-independent Fe uptake on neoplastic and normal cell growth and proliferation is discussed.

L15 ANSWER 36 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1984:83946 HCAPLUS
DN 100:83946
TI Effect of an anti-murine transferrin receptor-ricin A conjugate on bone marrow stem and progenitor cells treated in vitro
SO Exp. Cell Res. (1984), 150(2), 400-7
CODEN: ECREAL; ISSN: 0014-4827
AU Lesley, Jayne; Domingo, Derrick L.; Schulte, Roberta; Trowbridge, Ian S.

PY 1984
AB A monoclonal antibody with specificity for the murine transferrin receptor was conjugated with the toxic A subunit of ricin. The concn. range, specificity, and kinetics of inhibition of protein synthesis of the conjugate were detd. on the murine T-lymphoma cell line BW5147. When toxin was present throughout the period of culture, in vitro myeloid (CFUc) and erythroid (CFUe and BFUe) bone marrow colonies were inhibited by concns. of conjugate comparable to those that inhibit protein synthesis in murine cell lines. Bone marrow exposed briefly to antitransferrin receptor antibody-ricin A conjugate was assayed for myeloid and erythroid progenitors in vitro and for in vivo spleen colony formation. Only CFUe were depleted by this pulse exposure, consistent with the higher frequency of proliferating cells and transferrin receptor expression in the CFUe population relative to other progenitors.

L15 ANSWER 37 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1982:542886 HCAPLUS
DN 97:142886
TI Murine cell surface transferrin receptor: studies with an anti-receptor monoclonal antibody
SO J. Cell. Physiol. (1982), 112(3), 403-10
CODEN: JCLLAX; ISSN: 0021-9541
AU Trowbridge, Ian S.; Lesley, Jayne; Schulte, Roberta
PY 1982
AB A rat monoclonal antibody against the murine transferrin receptor was prepd. The receptor is a 95,000 mol. wt. species that exists in the cell membrane as a disulfide-bonded dimer. Whereas 29 of 29 urine hematopoietic tumor cell lines express detectable nos. of transferrin receptors, <1% of adult thymocytes or spleen cells and only 5% of bone marrow cells are pos. However, fetal liver and neonatal spleen contain substantial nos. of transferrin receptor-pos. cells. Induction of Friend cells in vitro with DMSO leads to an overall increase in the expression of transferrin receptors on the cell surface. The anti-transferrin receptor antibody partially blocks Fe uptake from 59Fe-transferrin by a variety of murine cell lines and inhibits the growth of a murine myeloma cell line in vitro.

L15 ANSWER 38 OF 38 HCAPLUS COPYRIGHT 1997 ACS
AN 1982:160566 HCAPLUS
DN 96:160566
TI A monoclonal antibody that detects expression of transferrin receptor in human erythroid precursor cells
SO Blood (1982), 59(3), 671-8
CODEN: BLOOAW; ISSN: 0006-4971
AU Lebman, Deborah; Trucco, Massimo; Bottero, Lisabianca; Lange, Beverly; Pessano, Silvana; Rovera, Giovanni
PY 1982
AB A monoclonal antibody, L5.1, obtained by immunizing a Balb/c mouse with HL60 human promyelocytic leukemia cells, reacts with both HL60 cells and with the K562(S) cell line. This monoclonal antibody binds and immunoppts. a glycoprotein (87,000 mol. wt.) present on the cell surface membrane of K562(S) as a disulfide bonded dimer. In competition expts., L5.1 competes with both transferrin and OKT9 (a known antitransferrin receptor antibody) for binding to target K562(S) erythroleukemia cells. Binding of both L5.1 and transferrin to the surface of K562(S) cells is inhibited by treatment with 12-O-tetradecanoyl-phorbol-13-acetate, and the extent and time course of inhibition is similar in both cases. L5.1 reacts strongly with all the morphol. recognizable human erythroid lineage precursors, from the pronormoblast to the orthochromatic normoblast, and with reticulocytes. Erythrocytes, myeloid elements, monocytes, megakaryocytes, platelets, peripheral blood B and T lymphocytes do not bind significantly with this antibody and only a small fraction of promyelocytes was reactive. Antibody L5.1 did not react with

leukemic cells of patients with acute lymphoblastic, myeloblastic, and promyelocytic leukemias, but it did react with some established B (1 of 5) and T (2 of 3) cell lines, and a myeloid (1 of 3) cell line, and with phytohemagglutinin-stimulated peripheral blood lymphocytes. The nonhemopoietic cells lines tested did not bind with L5.1 with the exception of a colorectal adenocarcinoma and a melanoma cell line, which were both strongly pos. The relation of antibody L5.1 to other monoclonal antibodies that bind the transferrin receptor is discussed.

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            ONAL)
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S9        77   RD (unique items)
S10       77   S9 AND S5
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 10/7/1     (Item 1 from file: 5)
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  In vivo redirection of immune cells against tumor using murine
hematopoietic stem cells retrovirally transduced with a chimeric
*monoclonal* *antibody*-T-cell *receptor* gene
  Wang G; Chopra R; Royal R; Yang J; Man Y; Zhuang Z; Rosenberg S; Hwu P
  National Cancer Inst., Bethesda, MD 20892, USA
  Proceedings of the American Association for Cancer Research Annual
Meeting 38 (0). 1997.  12.
  Full Journal Title:  Eighty-eighth Annual Meeting of the American
Association for Cancer Research, San Diego, California, USA, April 12-16,
1997.  Proceedings of the American Association for Cancer Research Annual
Meeting
  ISSN: 0197-016X
  Language: ENGLISH
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Print Number: Biological Abstracts/RRM Vol. 049 Iss. 006 Ref. 094266

10/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


13398093 BIOSIS Number: 99398093
Expression of receptor tyrosine kinases, KIT, TIE and HTK in hematopoietic progenitor cells
Sato A; Iwama A; Inada T; Hashiyama M; Suda T
Dep. Cell Differentiation, IMEG, Kumamoto Univ., Sch. Med., Kumamoto, Japan
Tissue Antigens 48 (4-2). 1996. 400.
Full Journal Title: 6th International Workshop and Conference on Human Leukocyte Differentiation Antigens, Kobe, Japan, November 10-14, 1996. Tissue Antigens
ISSN: 0001-2815
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 049 Iss. 003 Ref. 044732

10/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


13229130 BIOSIS Number: 99229130
Evidence of free and bound leptin in human circulation: Studies in lean and obese subjects and during short-term fasting
Sinha M K; Opentanova I; Ohannesian J P; Kolaczynski J W; Heiman M L; Hale J; Becker G W; Bowsher R R; Stephens T W; Caro J F
Thomas Jefferson Univ., Jefferson Med. Coll., 1025 Walnut St., Room 813 College Build., Philadelphia, PA 19107, USA
Journal of Clinical Investigation 98 (6). 1996. 1277-1282.
Full Journal Title: Journal of Clinical Investigation
ISSN: 0021-9738
Language: ENGLISH
Print Number: Biological Abstracts Vol. 102 Iss. 010 Ref. 144760

Little is known about leptin's interaction with other circulating proteins which could be important for its biological effects. Sephadex G-100 gel filtration elution profiles of 125Ileptin-serum complex demonstrated 125I-leptin eluting in significant proportion associated with macromolecules. The 125I-leptin binding to circulating macromolecules was specific, reversible, and displaceable with unlabeled leptin (ED-50: 0.73 +- 0.09 nM, mean +- SEM, n = 3). Several putative leptin binding proteins were detected by leptin-affinity chromatography of which either 80- or 100-kD proteins could be the soluble leptin receptor as apprx 10% of the bound 125I-*leptin* was immunoprecipitable with *leptin* *receptor* *antibodies*. Significantly higher (P lt 0.001) proportions of total leptin circulate in the bound form in lean (46.5 +- 6.6%) compared with obese (21.4 +- 3.4%) subjects. In lean subjects with 21% or less body fat, 60-98% of the total leptin was in the bound form. Short-term fasting significantly decreased basal leptin levels in three lean (P lt 0.0005) and three obese (P lt 0.005) subjects while refeeding restored it to basal levels. The effects of fasting on free leptin levels were more pronounced in lean subjects (basal vs. 24-h fasting: 19.6-1.9 vs. 1.3+-0.4 ng/ml) compared with those in obese subjects (28.3+-9.8 vs. 14.7-5.3). No significant (P gt 0.05) decrease was observed in bound leptin in either group. These studies suggest that in obese individuals the majority of leptin circulates in free form, presumably bioactive protein, and thus obese subjects are resistant to free leptin. In lean subjects with relatively low adipose tissue, the majority of circulating leptin is in the bound form and thus may not be available to brain receptors for its inhibitory effects on food intake both under normal and food deprivation states.

10/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


12122582 BIOSIS Number: 98722582
Immobilized anti-KIT monoclonal antibody induces ligand-independent dimerization and activation of steel factor receptor: Biologic similarity with membrane-bound form of steel factor rather than its soluble form
Kurosawa K; Miyazawa K; Gotoh A; Katagiri T; Nishimaki J; Ashman L K; Toyama K
First Dep. Intern. Med., Tokyo Medical College, 6-7-1, Nishishinjuku, Shinjuku-ku, Tokyo 160, Japan
Blood 87 (6). 1996. 2235-2243.
Full Journal Title: Blood
ISSN: 0006-4971
Language: ENGLISH
Print Number: Biological Abstracts Vol. 101 Iss. 008 Ref. 106857
Interaction of a tyrosine kinase type receptor and its ligand induces receptor-dimerization or -oligomerization followed by transphosphorylation and activation of its intrinsic kinase, which leads to a series of intracellular signals. We have previously reported that the membrane-bound form of Steel factor (SLF) induces more persistent tyrosine kinase activation and longer life span of c-kit encoded protein (KIT) than its soluble form (Miyazawa et al, Blood 85:641, 1995). In this study, we used YB5.B8 monoclonal antibody (MoAb) that recognizes the extracellular domain of KIT to investigate whether immobilized anti-KIT MoAb can substitute for SLF as a potent activator of KIT by cross-linking receptors and further compared its effect with each SLF isoform in a factordependent cell line MO7e. YB5.B8 MoAb in a soluble state suppressed SLF-induced MO7e cell proliferation in a dose-dependent manner. By contrast, once this antibody was immobilized on the goat-antimouse MoAb (GAM)-coated culture plates, it supported the growth of MO7e cells in the absence of any growth factors, whereas culture the cells in GAM alone or YB5.B8 without GAM-coated plates resulted in rapid cell-death within 24 hours. As with the natural ligand SLF, immobilized YB5.B8 MoAb synergized with granulocyte-macrophage colony-stimulating factor (GM-CSF) in inducing cell proliferation compared with either YB5.B8 MoAb or GM-CSF alone. Immunoblotting with antiphosphotyrosine MoAb showed that interaction of MO7e cells with immobilized YB5.BS induced tyrosine phosphorylation of a series of intracellular proteins including KIT (145 kD). In addition, cross-linking studies using a water-soluble crosslinking reagent bis-sulfosuccinimidyl-suberate showed that immobilized YB5.B8 MoAb induced dimerization and activation of KIT. However, as with stimulation by the membrane-bound form of SLF, the kinetics of KIT activation with YB5.B8 MoAb was more prolonged compared with the cells treated with recombinant soluble SLF. Flow cytometry showed that, unlike the cells treated with soluble SLF, no downmodulation of cell-surface KIT expression was observed in MO7e cells cultured with immobilized YB5.B8 MoAb. These data suggest that immobilized *antibodies* against *hematopoietic* *receptors* may replace their ligand-stimulators; however, their activities may resemble the membrane-bound form rather than the soluble form of natural ligands.

10/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


11665702 BIOSIS Number: 98265702
Insulin-dependent tyrosine phosphorylation of the vav proto-oncogene product in cells of hematopoietic origin
Uddin S; Katzav S; White M F; Platanias L C
Div. Hematol.-Oncol., Loyola Univ. Chicago, Build. 112, 2160 South First Ave., Maywood, IL 60153, USA
Journal of Biological Chemistry 270 (13). 1995. 7712-7716.
Full Journal Title: Journal of Biological Chemistry
ISSN: 0021-9258

Language: ENGLISH
Print Number: Biological Abstracts Vol. 099 Iss. 012 Ref. 170620
Insulin activates the ras signaling pathway and promotes hematopoietic cell proliferation. One possible mediator in such signaling is the vav proto-oncogene product (p95-vav), which is specifically expressed in cells of hematopoietic origin and contains domains typical of guanine nucleotide exchange factors as well as Src homology 2 and Src homology 3 domains. We studied the tyrosine phosphorylation of p95-vav in *hematopoietic* cells expressing insulin *receptors* . Immunoblotting experiments with an antiphosphotyrosine *monoclonal* antibody disclosed that insulin induces rapid and transient tyrosine phosphorylation of p95-vav in the human U-266 myeloma cell line. These findings were confirmed by immunoprecipitation experiments performed with 32P-labeled cells and phosphoamino acid analysis of the bands corresponding to p95-vav. Similarly, insulin-dependent tyrosine phosphorylation of p95-vav was observed in the human IM-9 and mouse J558L hematopoietic cell lines. Furthermore, insulin treatment of cells led to the association of the Src homology 2 domain of p95-vav with the activated beta-subunit of the insulin receptor in vitro. Altogether, these data suggest that p95-vav is a substrate for the insulin receptor tyrosine kinase and may be involved in an insulin signaling pathway linking receptor-generated signals to Ras or other GTP-binding proteins in cells of hematopoietic origin.

10/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


11307435 BIOSIS Number: 97507435
The c-kit proto-oncogene receptor is expressed on a subset of human CD3-CD4-CD8-(triple-negative) thymocytes
Decastro C M; Denning S M; Langdon S; Vandenbark G R; Kurtzberg J; Scearce B; Haynes B F; Kaufman R E
Box 3250, Duke Univ. Med. Cent., Durham, NC 27710, USA
Experimental Hematology (Charlottesville) 22 (10). 1994. 1025-1033.
Full Journal Title: Experimental Hematology (Charlottesville)
ISSN: 0301-472X
Language: ENGLISH
Print Number: Biological Abstracts Vol. 098 Iss. 011 Ref. 143174
The c-kit receptor is a tyrosine-kinase transmembrane receptor first identified as an oncogene in the HZ4-feline leukemia virus and later found to be important in hematopoiesis in mice. The ligand for this receptor (Steel factor) can stimulate hematopoiesis both in vitro and in vivo. To study the pattern of c-kit *receptor* expression in normal human *hematopoietic* progenitor cells, we prepared a *monoclonal* *antibody* (9B9) against human c-kit *receptor* by using a synthetic peptide (amino acids 476-501) from the extracellular domain of c-kit *receptor* to immunize Balb/c mice. *Monoclonal* antibody 9B9 bound to recombinant c-kit protein, the erythroleukemic line HEL, the megakaryocytic line MEG-01, and the murine mast cell line P815. Monoclonal antibody 9B9 also bound to the surface of the CD7+CD3-CD4-CD8- T cell lymphoid cell lines DU.528 and HSB2T, and also to 1 to 4% of normal bone-marrow cells. The majority (67 +- 6%) of CD34+ bone-marrow progenitor cells coexpressed c-kit receptor. Flow-cytometry analysis of immature CD3-CD4-CD8- (triple-negative) thymocytes indicated 30 +- 9.5% expressed the c-kit receptor, and thymidine incorporation assay revealed that the receptor is functional. Indirect fluorescent microscopy of human thymic tissue, using a monoclonal antibody against Steel factor, revealed its presence on scattered mononuclear cells within the intralobular septae and the subcapsular cortex, which are regions where the triple-negative thymocytes are also localized. These data provide evidence that the c-kit receptor is present on human hematopoietic bone marrow and intrathymic T cell progenitor cells, and that it likely plays a role in early T cell lymphopoiesis.

10/7/7 (Item 7 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)


10904614 BIOSIS Number: 97104614
Reactivity of the *monoclonal* *antibodies* from the cytokine *receptor* panel with normal *hemopoietic*, leukemic cells and cells of cell lines
Koubek K; Marinov I; Eckschlager T
Inst. Hematol. Blood Transfusion, Prague 2, CZR
Tissue Antigens 42 (4). 1993. 343.
Full Journal Title: 5th International Conference on Human Leukocyte Differentiation Antigens, Boston, Massachusetts, USA, November 3-7, 1993. Tissue Antigens
ISSN: 0001-2815
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 003 Ref. 033507

10/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


9605381 BIOSIS Number: 94110381
INHIBITION OF HEMATOPOIETIC TUMOR GROWTH BY COMBINED TREATMENT WITH DEFEROXAMINE AND AN IGM *MONOCLONAL* *ANTIBODY* AGAINST THE TRANSFERRIN *RECEPTOR* EVIDENCE FOR A THRESHOLD MODEL OF IRON DEPRIVATION TOXICITY
KEMP J D; THORSON J A; STEWART B C; NAUMANN P W
5238 CARVER, UNIVERSITY IOWA HOSPITALS, IOWA CITY, IOWA 52242.
CANCER RES 52 (15). 1992. 4144-4148. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
Recent studies have suggested that iron deprivation may represent a useful new approach in cancer therapy, and several strategies for producing such deprivation are now under investigation. Thus, for example, we recently provided evidence that combined treatment with the iron chelator deferoxamine and an IgG *monoclonal* *antibody* against the transferrin *receptor* (ATRA) produces synergistic inhibition of *hematopoietic* tumor cell growth in vitro (J. D. Kemp, K. M. Smith, L. J. Kanner, F. Gomez, J. A. Thorson, and P. W. Naumann, Blood, 76: 991-995, 1990). The current study is an attempt to analyze the mechanisms responsible for the synergistic interaction. The data show that a single IgG ATRA can produce up to 75% inhibition of iron uptake while having little effect on DNA synthesis; this suggests that tumor cells either take up or have stored amounts of iron well in excess of that required to suport immediate metabolic needs. When deferoxamine and the IgG ATRA are used together, the effects on iron acquisition and receptor down-modulation are either additive or subadditive but are clearly not synergistic. Overall, the findings suggest that the IgG ATRA produces an injury to iron uptake that is just below a critical threshold and that the additional effect provided by the iron chelator is sufficient to exceed that threshold and produce a rapid depletion of iron pools that are vital for short-term DNA synthesis. IgG ATRAS thus seem to be of even greater interest as therapeutic reagents, and further study of their properties and of how they interact with deferoxamine appears to be warranted.

10/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


8137041 BIOSIS Number: 91058041
MEGALOBLASTIC HEMATOPOIESIS IN-VITRO INTERACTION OF ANTI-FOLATE *RECEPTOR* *ANTIBODIES* WITH *HEMATOPOIETIC* PROGENITOR CELLS LEADS TO A PROLIFERATIVE RESPONSE INDEPENDENT OF MEGALOBLASTIC CHANGES
ANTONY A C; BRIDDELL R A; BRANDT J E; STRANEVA J E; VERMA R S; MILLER M E ; KALASINSKI L A; HOFFMAN R
DIVISION HEMATOLOGY/ONCOLOGY, DEP. MEDICINE, INDIANA UNIVERSITY SCHOOL

MEDICINE, INDIANAPOLIS, INDIANA 46202-5121.
J CLIN INVEST 87 (1). 1991. 313-325. CODEN: JCINA
Full Journal Title: Journal of Clinical Investigation
Language: ENGLISH

We tested the hypothesis that anti-placental folate receptor (PFR) antiserum-mediated effects on hematopoietic progenitor cells in vitro of increased cell proliferation and megaloblastic morphology were independent responses. We determined that (a) purified IgG from anti-PFR antiserum reacted with purified apo- and holo-PFR and specifically immunoprecipitated a single (44-kD) iodinated moiety on cell surfaces of low density mononuclear cells (LDMNC); (b) when retained in culture during in vitro hematopoiesis, anti-PFR IgG (in contrast in PFR-neutralized anti-PFR IgG and nonimmune IgG) consistently led to increased cloning efficiency of colony forming unit erythroid (CFU-E), burst forming unit-E (BFU-E), CFU-granulocytes macrophage (CFU-GM), and CFU-GEM, megakaryocyte (CFU-GEMM), and objectively defined megaloblastic changes in orthochromatic normoblasts from CFU-E- and BFU-E-derived colonies; (c) when anti-PFR antiserum was proved after initial (< 1 h) incubation with LDMNC, a cell proliferation response was induced, but megaloblastic changes were not evident. (d) Conversely, delay at 4.degree. C for 24 h before long-term plating with antiserum resulted in megaloblastosis without increased cell proliferations; (e) however, 500-fold molar excess extracellular folate concentrations completely abrogated the expected anti-PFR antiserum-induced megaloblastic changes, without altering cell proliferative responses. Thus, although cell proliferative and megaloblastic changes are induced after short-term and prolonged interaction of *antibody* with folate *receptors* on *hematopoietic* progenitors, respectively, there are independent effects.

10/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


6556487 BIOSIS Number: 86023038
IDENTIFICATION AND PURIFICATION OF HUMAN ERYTHROID PROGENITOR CELLS BY *MONOCLONAL* *ANTIBODY* TO THE TRANSFERRIN *RECEPTOR* TU 67
HERRMANN F; GRIFFIN J D; SABBATH K D; OSTER W; WERNET P; MERTELSMANN R
ABTEILUNG FUER HAEMATOLOGIE, I. MEDIZINISCHE KLINIK, JOHANNES GUTENBERG-UNIV., D-6500 MAINIZ, WEST GERMANY.
BLUT 56 (4). 1988. 179-184. CODEN: BLUTA
Full Journal Title: Blut
Language: ENGLISH

Anit-TU 67 is a murine *monoclonal* *antibody* that recognizes the transferrin *receptor*. With respect to *hematopoietic* cells TU 67 is expressed by human multipotent colony-forming cells (CFU-Mix), erythroid progenitor cells (BFU-E and CFU-E) and a fraction of granulocyte/monocyte colony forming cells, but is not expressed by mature hematopoietic cells including erythrocytes, platelets, lymphocytes, and peripheral blood myeloid cells. The TU 67-positive fraction of normal bone marrow, separated by fluorescence-activated cell sorting (FACS) or immune rosettes, contained 87% of the erythroid progenitor cells. Erythroid progenitor cells were enriched up to 50-fold by using a combination of monoclonal antibodies to deplete mature hematopoietic cells, followed by positive selection of BFU-E and CFU-E by TU 67 antibody.

10/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


6438672 BIOSIS Number: 85039193
SPECIFICITY OF A MOUSE MONOCLONAL ANTIBODY RAISED AGAINST ACUTE MYELOID LEUKEMIA CELLS FOR MAST CELLS IN HUMAN MUCOSAL AND CONNECTIVE TISSUES
MAYRHOFER G; GADD S J; SPARGO L D J; ASHMAN L K
DEP. MICROBIOL. AND IMMUNOL., UNIV. ADELAIDE, ADELAIDE, SOUTH AUSTRALIA

5001.
IMMUNOL CELL BIOL 65 (3). 1987. 241-250. CODEN: ICBIE
Full Journal Title: Immunology and Cell Biology
Language: ENGLISH
A mouse monoclonal antibody raised against acute myeloid leukaemia cells (YB5.B8 monoclonal antibody; Gadd, S. J. and Ashman, L. K. (1985): Leukaemia Res. 9, 1329-1336) has been found by an indirect immunoperoxidase technique to bind to scattered cells in frozen sections from a number of human tissues. They have been identified as mast cells in fixed sections of skin, tonsil and duodenum by simultaneous staining of glycosaminoglycan with Alcian blue in 0.7 N HCl. The antibody does not distinguish mast cells in mucosal tissues from those in connective tissue, although the level of expression by cells at both sites appears to be heterogeneous. With the exception of low affinity binding to B lymphocytes, no other bone marrow-derived cells were found to bind the antibody. In particular, basophils and esoinophils were not stained, suggesting that they are not related closely to mast cells and that the antigen detected by YB5.B8 monoclonal *antibody* is not an IgE Fc *receptor*. Therefore, among all mature *haemopoietic* lineages, the *antibody* is specific for mast cells.

10/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


6211520 BIOSIS Number: 35077041
OSTEOCLAST-SPECIFIC ANTIGENS
HORTON M A
I.C.R.F. HAEMOPOIESIS RES. GROUP, DEP. HAEMATOL., MED. COLL. ST. BARTHOLOMEW'S HOSP., LONDON EC1A 7BE, U.K.
ISI ATLAS SCI IMMUNOL 1 (1). 1988. 35-43. CODEN: IASIE
Full Journal Title: Isi Atlas of Science Immunology
Language: ENGLISH

10/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


5344508 BIOSIS Number: 81111815
A RADIOIMMUNOASSAY THAT SANDWICHES HUMAN INTERLEUKIN 2 BETWEEN RADIOLABELED *MONOCLONAL* *ANTIBODY* AND THE *RECEPTOR* ON A *HEMATOPOIETIC* CELL LINE
OHIKE Y; IMAI M; TANAKA E; MUKAIDA N; KASAHARA T; TACHIBANA K; MIYAKAWA Y ; MAYUMI M
IMMUNOLOGY DIVISION, JICHI MEDICAL SCHOOL, MINAMIKAWACHI-MACHI, TOCHIGI-KEN 329-04, JAPAN.
J IMMUNOL METHODS 87 (2). 1986. 245-250. CODEN: JIMMB
Full Journal Title: Journal of Immunological Methods
Language: ENGLISH
Two monoclonal antibodies were raised against human interleukin-2 (IL-2) produced by Escherichia coli harboring recombinant complemental DNA. Both antibodies did not neutralize its activity, nor did they inhibit the binding of IL-2 to the receptor on target cells. Taking advantage of the ability of monoclonal antibodies to detect IL-2 that had bound to the receptor, a radioimmunoassay was developed that sandwiched IL-2 between the radiolabeled *monoclonal* *antibody* and the *receptor* on a *hematopoietic* cell line infected with human T cell leukemia virus Type I. The assay had the advantage of detecting only IL-2 with the ability to bind to the receptor, and displayed a linear dose-response relationship over concentrations ranging from 5 to 100 ng/ml.

10/7/14 (Item 1 from file: 35)
DIALOG(R)File 35:Dissertation Abstracts Online

01557036 ORDER NO: AAD97-17012
RESCUE FROM APOPTOSIS IN EARLY (CD34 SELECTED) VERSUS LATE (CD34 UNSELECTED) HUMAN HEMATOPOIETIC CELLS BY VLA-4 AND VCAM-1 DEPENDENT ADHESION TO BONE MARROW STROMAL CELLS
Author: WANG, WEN-JUNG MICHAEL
Degree: PH.D.
Year: 1997
Corporate Source/Institution: THE UNIV. OF TEXAS H.S.C. AT HOUSTON GRAD. SCH. OF BIOMED. SCI. (2034)
Supervisor: ALBERT B. DEISSEROTH
Source: VOLUME 57/12-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 7442. 106 PAGES

The interaction of hematopoietic precursor cell with bone marrow stromal cells is assumed to be important to the survival of hematopoietic precursor cells during hematopoietic cell long-term culture. Early hematopoietic stem cells are preferentially found within the stromal adherent cell fraction in primary long-term bone marrow cultures. The purpose of this dissertation was to understand the molecular mechanisms that govern these interactions for the regulation of survival and proliferation of early versus late hematopoietic cells.

Monoclonal antibodies to the VLA-4 recognize the alpha4 beta1 integrin *receptor* on human *hematopoietic* cells. This *monoclonal* *antibody* blocks the adhesion between early hematopoietic progenitor cells (CD34 selected cells) and stromal cells when added to cultures of these cells. Addition of the VLA-4 monoclonal antibody to cultures of stromal cells and CD34 selected cells was shown to induce apoptosis of CD34 selected cells in these CD34 selected cell/stromal cell cocultures, as measured by the terminal deoxynucleotidyl transferase (TdT)-mediated dUTP-biotin nick end-labeling method. In contrast to these experiments with early hematopoietic progenitor cells (CD34+), the level of adhesion between more differentiated cells (unfractionated hematopoietic cells) and stromal cells was not significantly altered by addition of the anti-VLA-4 monoclonal antibody. Similarly, the level of apoptosis of unfractionated hematopoietic cells was not significantly increased by the addition of anti-VLA-4 monoclonal antibody to cultures of the latter cells with stromal cells. The binding of the unfractionated cells is less than that of the CD34 selected. Since there is no difference between the alpha4 beta1 integrin expression level of the early and late myeloid cells, there may be a difference in the functional state of the integrin between the early and late myeloid cells. We also show that CD34+ selected precursor cells proliferate at a higher rate when these cells are plated on recombinant VCAM-1 molecules. These data indicate that the alpha4beta1 integrin receptor (VLA-4) plays a central role in the apoptosis rescue function which results from the anchorage-dependent growth of the CD34 selected early hematopoietic cells on stromal cells. The data suggest that these apoptosis rescue pathways have less significance as the cells mature and become anchorage-independent in their growth. These data should assist in the design of systems for the ex vivo proliferation and transduction of early hematopoietic cells for genetic therapy.

10/7/15 (Item 1 from file: 65)
DIALOG(R)File 65:Inside Conferences


01009329 INSIDE CONFERENCE ITEM ID: CN009867934
Reactivity of Cytokine *Receptor* Panel *mAb* with immature *haemopoietic* cells and cell lines
Buehring, H.-J.; Burkhardt, M.; Ning, Y.; Zhu, X.

Leucocyte typing V: white cell differentiation antigens
LEUCOCYTE TYPING, 1995; VOL 5//V2, P: 1945-1948
Oxford University Press, 1995
ISBN: 0192626892; 0192623761

LANGUAGE: English DOCUMENT TYPE: Conference Papers

CONFERENCE:
Leucocyte typing V: white cell differentiation antigens-5th International conference on human leukocyte differentiation antigens ( Leucocyte typing 5; Leucocyte typing five)
EDITOR(S): Schlossman, S. F.
LOCATION: Boston, MA
DATE: Nov 1993 (199311)
NOTE:
In 2 vols

10/7/16 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE


8581605 EMBASE No: 92257516
Inhibition of hematopoietic tumor growth by combined treatment with deferoxamine and an IgG *monoclonal* *antibody* against the transferrin *receptor*: Evidence for a threshold model of iron deprivation toxicity
Kemp J.D.; Thorson J.A.; Stewart B.C.; Naumann P.W.
5238 Carver, University of Iowa Hospitals, Iowa City, IA 52242 USA
CANCER RES. (USA) , 1992, 52/15 (4144-4148) CODEN: CNREA ISSN: 0008-5472
LANGUAGES: English SUMMARY LANGUAGES: English
Recent studies have suggested that iron deprivation may represent a useful new approach in cancer therapy, and several strategies for producing such deprivation are now under investigation. Thus, for example, we recently provided evidence that combined treatment with the iron chelator deferoxamine and an IgG *monoclonal* *antibody* against the transferrin *receptor* (ATRA) produces synergistic inhibition of *hematopoietic* tumor cell growth in vitro (J. D. Kemp, K. M. Smith, L. J. Kanner, F. Gomez, J. A. Thorson, and P. W. Naumann, Blood, 76: 991-995, 1990). The current study is an attempt to analyze the mechanisms responsible for the synergistic interaction. The data show that a single IgG ATRA can produce up to 75% inhibition of iron uptake while having little effect on DNA synthesis; this suggests that tumor cells either take up or have stored amounts of iron well in excess of that required to support immediate metabolic needs. When deferoxamine and the IgG ATRA are used together, the effects on iron acquisition and receptor down-modulation are either additive or subadditive but are clearly not synergistic. Overall, the findings suggest that the IgG ATRA produces an injury to iron uptake that is just below a critical threshold and that the additional effect provided by the iron chelator is sufficient to exceed that threshold and produce a rapid depletion of iron pools that are vital for short-term DNA synthesis. IgG ATRAS thus seem to be of even greater interest as therapeutic reagents, and further study of their properties and of how they interact with deferoxamine appears to be warranted.

10/7/17 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE


8341226 EMBASE No: 92017484
Lipid metabolism and hyperandrogenism
Wild R.A.
Prevention Center, University of Oklahoma Health Sciences Center, Post Office Box 26901, 4SP-717, Oklahoma City, OK 73190 USA
CLIN. OBSTET. GYNECOL. (USA) , 1991, 34/4 (864-871) CODEN: COGYA ISSN: 0009-9201
LANGUAGES: English SUMMARY LANGUAGES: English
Hyperandrogenism and lipid metabolism were shown to be related intimately. Any discussion of the nature of their relationship must include other clinical and metabolic variables such as hyperinsulinemia and UBO.

Despite the many correlations among each of these factors, the appropriate sequence in the pathogenesis of these conditions has not been defined. Do conditions that result in insulin resistance (e.g., genetic defects, insulin *receptor* *antibodies*, and *obesity* ) also lead to the development of hyperandrogenemia by direct or indirect ovarian stimulation by insulin? Does hyperandrogenism of ovarian or adrenal origin cause abnormal upper body fat distribution, in turn leading to lipid abnormalities and insulin resistance? Regardless of the issue of mechanism of causality, women with hyperandrogenism are thought to be at greater risk for cardiovascular morbidity and mortality than their normoandrogenic counterparts. These women often are obese, hypertensive, and sedentary; ingest diets high in saturated fats; and have glucose intolerance and/or insulin resistance. All these abnormalities are well known independent risk factors for the development of lipid abnormalities and cardiovascular disease. Whether hyperandrogenism is a secondary consequence of any of these or whether it is an independent contributor to lipid aberrations requires future study. Treatment strategies for hyperandrogenic women, however, should not only be directed toward alleviation of the cosmetic problem of hirsutism but also toward the prevention and treatment of cardiovascular morbidity using modalities aimed at eradicating hyperinsulinemia, hypertension, and dyslipidemia. These modalities should include modifications in diet, exercise, and weight in addition to pharmacologic and/or surgical manipulation. Weight reduction will reduce many cardiovascular risk factors. Obesity is easier to target because of the many risk factors that result in it. Whether treatment of hyperandrogenemia per se can contribute to reduced cardiovascular risk remains to be determined.

10/7/18 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE


7776500 EMBASE No: 90204476
Specific inhibition of interleukin 3 bioactivity by a monoclonal antibody reactive with hematopoietic progenitor cells
Emanuel P.D.; Peiper S.C.; Chen Z.; Sheng D.C.; Zuckerman K.S.
Division of Hematology/Oncology, University of Alabama, 513 Tinsley Harrison Tower, Birmingham, AL 35294 USA
PROC. NATL ACAD. SCI. U. S. A. (USA) , 1990, 87/12 (4449-4452) CODEN: PNASA ISSN: 0027-8424
LANGUAGES: English
HIM1, originally designated HI98, a murine monoclonal IgM antibody raised against human mononuclear cells, has been reported at the Fourth International Leukocyte Typing Workshop (called antibody M0141) to be the only one of 157 antibodies tested that inhibited binding of interleukin 3 (IL-3) to KG-1 human acute myelogenous leukemia cells and normal human monocytes. We have carried out detailed studies of the selective effect of HIM1 on IL-3-mediated stimulation of hematopoietic progenitors. Preincubation of normal human bone marrow mononuclear cells, depleted of adherent cells and T cells, with HIM1 antibody resulted in a dose-dependent inhibition of IL-3-mediated stimulation of both erythroid burst-forming units (maximum inhibition 55%) and granulocyte/macrophage colony-forming units (maximum inhibition 49%). HIM1 antibody had no effect on growth of erythroid colony-forming units in culture. In addition, preincubation of the cells with HIM1 antibody had no deleterious effect on granulocyte/macrophage colony-stimulating factor-induced growth of either erythroid bursts or granulocyte/macrophage colonies. To be certain that the HIM1 antibody did not react directly with IL-3 itself, we attempted to use immunodepletion to remove IL-3 that had been added to our culture medium. Although we were able to remove IL-3 bioactivity by immunodepletion with anti-IL-3 antibody bound to Sepharose beads, beads with attached HIM1 did not remove IL-3 activity from the medium. Polymorphonuclear neutrophils bind high levels of HIM1, although they have very few or no detectable IL-3 *receptors*. Therefore, this *antibody* appears to recognize a cell surface antigen that is critical for optimal IL-3 binding and bioactivity but is

not the actual IL-3 receptor.

10/7/19 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE


7281457 EMBASE No: 88181785
Insulin receptor defects as cause of disease
INSULINREZEPTORDEFEKTE ALS KRANKHEITSURSACHE
Dreyer M.; Rudiger H.W.
Krankenhaus Bethanien, D-2000 Hamburg 20 Germany, Federal Republic of
INTERNIST (Germany, Federal Republic of) , 1988, 29/6 (390-396) CODEN: INTEA ISSN: 0020-9554
LANGUAGES: German

10/7/20 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE


6239704 EMBASE No: 86234767
Membrane IgM, IgD, and IgG act as signal transmission molecules in a series of B lymphomas
Mizuguchi J.; Tsang W.; Morrison L.; et al.
Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, The National Institutes of Health, Bethesda, MD 20892 USA
J. IMMUNOL. (USA) , 1986, 137/7 (2162-2167) CODEN: JOIMA
LANGUAGES: ENGLISH
Increases in intracellular free calcium concentration ((Casup 2sup +)(i)) were observed in response to anti-immunoglobulin (Ig) antibodies in each of six B cell tumors or B cell hybridomas bearing mu or delta chains on their cell surface. The BAL17 cell line, bearing mu and delta chains on its surface, behaved similarly to mature B cells in the following respects. Anti-IgM and anti-IgD antibodies caused increases in (Casup 2sup +)(i) and inositol phospholipid metabolism; the initial increases in (Casup 2sup +)(i) were derived partly from an intracellular Casup 2sup + pool; lipopolysaccharide, phorbol myristate acetate (PMA), B cell stimulatory factor-1, and antibodies to class I and class II major histocompatibility molecules and to the Fcgamma receptor failed to cause increases in (Casup 2sup +)(i) or in inositol phospholipid metabolism; and increases in (Casup 2sup +)(i) and inositol phospholipid metabolism in response to anti-Ig were inhibited by pretreatment with PMA. Furthermore A20, an IgG2a bearing lymphoma, showed increases in (Casup 2sup +)(i) in response to anti-IgG2a, and a lymphoma cell line (6G8-2E10) expressing membrane IgG2b as a result of DNA-mediated transfer of the gamma(2b) H chain gene, showed increases in (Casup 2sup +)(i) in response to anti-IgG2b. These results indicate that Ig-bearing lymphomas display early events in B cell activation after receptor cross-linkage and can be used for detailed studies of the activation process.

10/7/21 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE


6197146 EMBASE No: 86192207
Disulphide-linked receptors for IgE on rat basophilic leukaemia cells
Roth P.A.; Rao M.; Froese A.
MRC Group in Allergy Research, Department of Immunology, University of Manitoba, Winnipeg, Man. R3E 0W3 CANADA
IMMUNOLOGY (ENGLAND) , 1986, 58/4 (671-676) CODEN: IMMUA
LANGUAGES: ENGLISH
Rat basophilic leukemia (RBL) cells are known to posses three different kinds of receptors capable of interacting with IgE, which have been named R, H and 71K and which differ in apparent molecular weight (MW) as

determined by SDS-PAGE. Reduction of receptors isolated from surface-iodinated RBL cells reduced the MW of 71K to a value corresponding to the MW of R without altering significantly the MW of H or R. Only a single reduction product of 71K could be detected when either surface-iodinated or sup 3H-leucine labelled RBL cells were used. Analysis of one- and two-dimensional tryptic peptide maps derived from surface-iodinated receptors revealed a similarity between R and 71K. The tryptic peptides of H exhibited an entirely different pattern. These results suggest that 71K consists either of two disulphide-linked R-like molecules, or of one such molecule linked to another as yet undetected polypeptide chain.

10/7/22 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE


5953582 EMBASE No: 85199092
The insulin receptor
Lyen K.R.
Department of Paediatrics, National University of Singapore, Singapore 0316 SINGAPORE
ANN. ACAD. MED. SINGAPORE (SINGAPORE) , 1985, 14/2 (364-373) CODEN: AAMSC
LANGUAGES: ENGLISH
The insulin receptor is a glycoprotein with a molecular weight in the order of 300,000. There are probably two pairs of subunits joined together by disulphide bonds. The distribution of receptors appears to be tissue-specific. On liver plasma membranes they are found predominantly as singletons, whereas on adipocytes they occur mainly in groups. The groups of receptors are held together by disulphide bonds, but these are different from the bonds holding the subunits together. When insulin binds to the receptor, the hormone-receptor complex is internalised in pinocytotic invaginations in the adipocyte, and in coated pits in fibroblasts. Half the receptors are transported to lysosomes where they are degraded, and the other half are recycled to the cell surface presumably for the further re-utilisation. Obese patients and those with type II diabetes have in common both a reduced number of insulin receptors and a post-receptor defect. However the degree of insulin resistance in type II diabetes cannot be accounted for on the basis of obesity alone. Moreover many types II diabetics are not obese. The insulin receptor is also altered in certain physiological states. Fasting and exercise lead to increased binding of insulin to its receptor. Pregnancy, on the other hand, may either increase or reduce binding. The effects of glucocorticoids are heterogeneous, and it is probable that the insulin resistance they induce is post-*receptor* in nature. Auto-*antibodies* to the insulin *receptor* is a rare cause of severe insulin-resistant diabetes, but the condition has given considerable insight into the nature of the insulin receptor. The insulin receptor appears to play a central role in determining the ensuing biological responses. What controls the receptor and modulates its function is likely to influence its actions. Precisely how insulin translates its message into biochemical actions is still incompletely understood.

10/7/23 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE


332381 EMBASE No: 75125310
Cyclic adenosine monophosphate and clinical medicine. Part II: Carbohydrate and lipid metabolism
Keirns J.J.; Freeman J.; Bitensky M.W.
Dept. Pathol., Yale Univ. Sch. Med., New Haven, Conn. 06510 USA
AMER.J.MED.SCI. (USA) , 1974, 268/2 (62-91) CODEN: AJMSA
LANGUAGES: ENGLISH
The molecular mechanism of insulin action is still not well understood,

certainly in comparison with the mechanism of glucagon and other peptide hormones whose direct interaction with adenyl cyclase has been repeatedly demonstrated. However, there is accumulating evidence that insulin influences cyclic nucleotide metabolism, diminishing cAMP levels and perhaps elevating cGMP levels. Great progress has been made in understanding the factors which regulate insulin secretion. One of the most important of these factors is the level of cAMP in the pancreatic beta cell. Peptide hormones, intestinal factors, and hypoglycemic sulfonylureas all promote insulin secretion by increasing the cAMP concentration in the beta cell. Most of the features of the acute diabetic state can be understood in terms of the absolute or relative deficiency of insulin, which translates biochemically into elevated concentrations of cAMP in liver, muscle, and fat. On the other hand, most types of hypoglycemia are caused by diminished cAMP levels (especially in the liver). Insulin resistance can be caused by failure of insulin to reach or bind to its target or by excessive amounts of a hormone which antagonizes insulin by elevating cAMP levels in the target cell, so that in either case the usual amounts of insulin fail to produce the required decreases in cAMP levels. Many details concerning the role of cAMP and of other metabolic and endocrine factors in the regulation of triglyceride deposition and mobilization have been elucidated. It is clear that cAMP is the major determinant in lipid mobilization and that the adipocyte adenyl cyclase cascade constitutes one of several metabolic loci whose malfunction may significantly impair lipid mobilization.

10/7/24 (Item 1 from file: 98)
DIALOG(R)File 98:Wilson General Sci Full-Text


02797470 H.W. WILSON RECORD NUMBER: BGSI94047470
Cloning of the cDNA for a hematopoietic cell-specific protein related to CD20 and the b subunit of the high-affinity IgE receptor: evidence for a family of proteins with four membrane-spanning regions.
Adra, Chaker N
Lelias, Jean-Michel; Kobayashi, Hirofumi
Proceedings of the National Academy of Sciences of the United States of America (Proc Natl Acad Sci U S A) v. 91 (Oct. 11 '94) p. 10178-82
LANGUAGE: English
COUNTRY OF PUBLICATION: United States

ABSTRACT: The cloning of the cDNA for a human gene, HTm4, that is specifically expressed in hematopoietic cells is reported. HTm4 protein shares about 20 percent sequence homology with the B-cell-specific antigen CD20 and with the b subunit of the high-affinity receptor for IgE (FceRIb); moreover, all 3 map to the same chromosomal location, 11q12-13.1. It is suggested that the HTm4, CD20, and FceRIb genes evolved from a common ancestral gene, giving rise to a family of 4-transmembrane proteins.

10/7/25 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal


13065230 PASCAL No.: 97-0355793
Expression and function of murine receptor tyrosine kinases, TIE and TEK, in hematopoietic stem cells
YANO M; IWAMA A; NISHIO H; SUDA J; TAKADA G; SUDA T
Department of Cell Differentiation, Institute of Molecular Embriology and Genetics (IMEG), Kumamoto University School of Medicine, Kumamoto, Japan; Department of Pediatrics, Akita University School of Medicine, Akita, Japan
Journal: Blood, 1997, 89 (12) 4317-4326
ISSN: 0006-4971 Availability: INIST-3178; 354000062000350080
No. of Refs.: 32 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: United States

Language: English
Two highly related receptor tyrosine kinases, TIE and TEK, comprise a family of endothelial cell-specific kinase. We established monoclonal antibodies against them and performed detailed analyses on their expression and function in murine hematopoietic stem cells (HSCs). TIE and TEK were expressed on 23.7% and 33.3% of lineage marker-negative, c-Kit SUP + and Sca-1 SUP + (Lin SUP - c-Kit SUP + Sca-1 SUP + ) HSCs that contain the majority of day-12 colony-forming units-spleen (CFU-S) and long-term reconstituting cells, but not committed progenitor cells. Lin SUP - c-Kit SUP + Sca-1 SUP + cells were further divided by the expression of TIE and TEK. TIE SUP + and TEK SUP + HSCs as well as each negative counterpart contained high proliferative potential colony-forming cells and differentiated into lymphoid and myeloid progenies both in vitro and in vivo. However, day-12 CFU-S were enriched in TIE SUP + and TEK SUP + HSCs. Our findings define TIE and TEK as novel stem cell marker antigens that segregate day-12 CFU-S, and provide evidence of novel signaling pathways that are involved in the functional regulation of HSCs at a specific stage of differentiation, particularly of day-12 CFU-S.



10/7/26 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal


10295548 PASCAL No.: 92-0501487
Inhibition of hematopoietic tumor growth by combined treatment with deferoxamine and an IgC *monoclonal* *antibody* against the transferrin *receptor* : evidence for a threshold model of iron deprivation toxicity
KEMP J D; THORSON J A; STEWART B C; NAUMANN P W
Univ. Iowa coll. medicine, dep. pathology, Iowa City IA 52242, USA
Journal: Cancer research : (Baltimore), 1992, 52 (15) 4144-4148
ISSN: 0008-5472 CODEN: CNREA8 Availability: INIST-5088; 354000020321430140
No. of Refs.: 19 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: USA
Language: English
Recent studies have suggested that iron deprivation may represent a useful new approach in cancer therapy, and several strategies for producing such deprivation are now under investigation. Thus, for example, we recently provided evidence that combined treatment with the iron chelator deferoxamine and an IgG *monoclonal* *antibody* against the transferrin *receptor* (ATRA) produces synergistic inhibition of *hematopoietic* tumor cell growth in vitro (J. D. Kemp, K. M. Smith, L. J. Kanner, F. Gomez, J. A. Thorson, and P. W. Naumann, Blood, 76: 991-995, 1990)

10/7/27 (Item 3 from file: 144)
DIALOG(R)File 144:Pascal


10230639 PASCAL No.: 92-0436542
Human monoclonal antibody detectsl a cell surface antigen expressed on hematopoietic malignant cells of lymphoid lineage
IIZASA T; YAMAGUCHI Y; TAGAWA M; FUJISAWA T; SAITO H; KONDO H; MATSUO Y; MONOWADA J; TANIGUCHI M
Chiba univ., inst. pulmonary cancer res., dep. surgery, 1-8-1 Inohana, Chiba 280, Japan
Journal: Japanese journal of cancer research : (Gann), 1991, 82 (2) 213-218
ISSN: 0910-5050 CODEN: GANNA2 Availability: INIST-2432; 354000017447710130
No. of Refs.: 23 ref.
Document Type: P (Serial) ; A (Analytic)

Country of Publication: Japan
Language: English

10/7/28 (Item 4 from file: 144)
DIALOG(R)File 144:Pascal


09640885 PASCAL No.: 91-0438008
Adhesion receptors involved in the erythrobalstic Island. Discussion
CROCKER P R; MORRIS L; GORDON S; COULOMBEL; CROCKER; ALLEN
Univ. Oxford, William Dunn school pathology, Oxford, United Kingdom
1Inst. Pasteur, Paris 75724, France
Journal: Blood cells, 1991, 17 (1) 83-96
ISSN: 0340-4684 CODEN: BLCEDD Availability: INIST-16766;
354000019304200070/NUM; INSERM-436
No. of Refs.: 24 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: Federal Republic of Germany
Language: English

10/7/29 (Item 5 from file: 144)
DIALOG(R)File 144:Pascal


05728322 PASCAL No.: 84-0229198
Expression of transferrin receptor on murine hematopoietic progenitors
LESLEY J; HYMAN R; SCHULTE R; TROTTER J
Salk inst., San Diego CA 92138, USA
Journal: Cellular Immunology, 1984, 83 (1) 14-25
ISSN: 0008-8749 Availability: CNRS-15106
No. of Refs.: 30 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: USA
Language: English

10/7/30 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


09198039 97364760
Ligand-independent dimerization of the extracellular domain of the leptin receptor and determination of the stoichiometry of leptin binding.
Devos R; Guisez Y; Van der Heyden J; White DW; Kalai M; Fountoulakis M; Plaetinck G
Roche Research Gent, F. Hoffmann-La Roche & Co., B-9000 Gent, Belgium.
J Biol Chem (UNITED STATES) Jul 18 1997, 272 (29) p18304-10, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The leptin receptor is a class I transmembrane protein with either a short or a long cytoplasmic domain. Using chemical cross-linking we have analyzed the binding of leptin to its receptor. Cross-linking of radiolabeled leptin to different isoforms of the leptin receptor expressed on COS-1 cells reveals leptin receptor monomer, homodimer, and oligomer complexes. Cotransfection of the long and short form of the leptin receptor did not provide any evidence for the formation of heterodimer complexes. Soluble forms consisting of either the entire extracellular domain or the two cytokine receptor homologous domains of the leptin receptor were purified to homogeneity from recombinant baculovirus-infected insect cells by leptin affinity chromatography. Gel filtration chromatography showed that these proteins exist in a dimeric form. Analysis of the complex formed between soluble leptin receptor and leptin by native polyacrylamide gel electrophoresis, and data obtained from the amino acid composition of the

complex provide direct evidence that the extracellular domain of the leptin receptor binds leptin in a 1:1 ratio.

10/7/31 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


08621543 96270476
Activation of beta(3) adrenergic receptors suppresses leptin expression and mediates a leptin-independent inhibition of food intake in mice.
Mantzoros CS; Qu D; Frederich RC; Susulic VS; Lowell BB; Maratos-Flier E; Flier JS
Division of Endocrinology, Beth Israel Hospital, Boston, Massachusetts, USA.
Diabetes (UNITED STATES) Jul 1996, 45 (7) p909-14, ISSN 0012-1797 Journal Code: E8X
Contract/Grant No.: P30DK462000, DK, NIDDK; K08 HL-02564, HL, NHLBI; K08 DK-02119, DK, NIDDK
Languages: ENGLISH
Document type: JOURNAL ARTICLE
To examine potential interactions between leptin and the beta3 adrenergic system in the regulation of food intake, we determined the effects of treatment with a selective beta3 adrenergic receptor (AR) agonist (CL 316,243 [1 mg/kg]) on body weight, food intake, and leptin expression. Studies were carried out in C57Bl/6J and FVB male control mice as well as in mice with targeted disruption of the beta3 AR gene. These findings were correlated with measurement of the expression in hypothalamus of neuropeptide Y (NPY) and melanin concentrating hormone (MCH), two neuropeptides that may be involved in the central regulation of food intake. Treatment with CL 316,243 (1 mg/kg) for 12 or 24 h decreased leptin mRNA abundance and circulating levels to 20% of baseline in normal animals. No effect of the CL 316,243 compound was seen in mice with targeted disruption of the beta3 AR gene. Despite the failing leptin levels, beta3 agonist administration acutely suppressed food intake. Finally, the induced suppression of food intake and leptin levels occurred despite unchanged or increased hypothalamic expression of the orexigenic neuropeptides NPY and MCH. Thus, beta3 AR agonists via beta3 ARs suppress leptin levels acutely and simultaneously suppress food intake via a mechanism that operates downstream of leptin and two of its putative central targets.

10/7/32 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


08393962 95112336
The fourth immunoglobulin domain of the stem cell factor receptor couples ligand binding to signal transduction.
Blechman JM; Lev S; Barg J; Eisenstein M; Vaks B; Vogel Z; Givol D; Yarden Y
Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.
Cell (UNITED STATES) Jan 13 1995, 80 (1) p103-13, ISSN 0092-8674 Journal Code: CQ4
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Receptor dimerization is ubiquitous to the action of all receptor tyrosine kinases, and in the case of dimeric ligands, such as the stem cell factor (SCF), it was attributed to ligand bivalency. However, by using a dimerization-inhibitory *monoclonal* *antibody* to the SCF *receptor*, we confined a putative dimerization site to the nonstandard fourth immunoglobulin-like domain of the receptor. Deletion of this domain not only abolished ligand-induced dimerization and completely inhibited signal transduction, but also provided insights into the mechanism of the coupling of ligand binding to dimer formation. These results identify an intrinsic

receptor dimerization site and suggest that similar sites may exist in other receptors.

10/7/33 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


08393193 95097248
Switching of mouse spermatogonial proliferation from the c-kit receptor-independent type to the receptor-dependent type during differentiation.
Tajima Y; Sawada K; Morimoto T; Nishimune Y
Research Institute for Microbial Diseases, Osaka University, Japan.
J Reprod Fertil (ENGLAND) Sep 1994, 102 (1) p117-22, ISSN 0022-4251
Journal Code: JWN
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Testicular cells composed mostly of germ cells and immature Sertoli cells from neonatal mice 2 and 5 days old were cultured to investigate germ-cell proliferation mediated by the c-kit *receptor*. The addition of *antibody* to block the interaction of the c-kit receptor with its ligand inhibited the proliferation of cultured spermatogonia from 5-day-old mice in a dose-dependent manner, but not from that of 2-day-old mice. The addition of anti-c-kit ACK2 monoclonal antibody also inhibited the proliferation of spermatogonia from 5-day-old mutant Sld/Sld mice but not of 5-day-old mutant Wv/Wv mice. The results indicate that c-kit-positive type A spermatogonia in the testes of 5-day-old mice require steel factor (kit ligand) for their proliferation, whereas self-renewal and differentiation of c-kit-negative primitive type A spermatogonia in the testes of 2-day-old mice do not.

10/7/34 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


08380780 94229206
A murine stromal cell line promotes the proliferation of the human factor-dependent leukemic cell line UT-7.
Auffray I; Dubart A; Izac B; Vainchenker W; Coulombel L
INSERM U 362, Institut Gustave Roussy, Villejuif, France.
Exp Hematol (UNITED STATES) May 1994, 22 (5) p417-24, ISSN 0301-472X
Journal Code: EPR
Languages: ENGLISH
Document type: JOURNAL ARTICLE
In long-term human bone marrow cultures, stromal cells of human origin are usually used on the assumption that human primitive progenitor cells do not respond to cytokines produced by stromal cells from other species. There is accumulating evidence, however, that murine stromal cells also promote maintenance and differentiation of very primitive human stem cells, which suggests the existence of novel stromal activities that cross species barriers. In this study, we show that a murine bone marrow-derived stromal cell line, MS-5, allows the proliferation of the human leukemic cell line UT-7. The long-term growth of UT-7 is usually supported only by human interleukin-3 (IL-3), granulocyte-macrophage colony-stimulating factor (GM-CSF), or erythropoietin (Epo). None of these three cytokines was involved in the observed effect, since murine GM-CSF and IL-3 do not act on human cells and MS-5 cells do not produce Epo. Soluble stem cell factor (SCF) induced UT-7 cell proliferation. However, Sl/Sl mutant fibroblasts also supported UT-7 cell growth and anti-c-kit antibodies only partially abolished UT-7 cell proliferative response to MS-5 cells. These observations excluded a major role of SCF in this system. MS-5-derived growth-promoting activity was diffusible, but attempts to grow UT-7 cells in high levels of known soluble murine stromal-derived cytokines active on human cells showed no or minimal response, suggesting that MS-5's

proliferative effect was not mediated by known cytokines. Finally, involvement of an autocrine loop of activation induced by MS-5 was excluded: RT-PCR analysis did not detect increased transcripts for GM-CSF, IL-3, IL-6, SCF, or Epo in UT-7 cells cocultured for 2 to 6 days with MS-5. In addition, UT-7 cell proliferation on MS-5 was not inhibited by neutralizing *antibodies* against the human GM-CSF *receptor* or the human IL-6 receptor alpha chain. Whether UT-7 cell proliferation triggered by MS-5 reflects the existence of novel stromal cytokines or results from synergistic interactions on the MS-5 cell surface between extracellular matrix proteins and cytokines will require further investigation.

10/7/35 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


07831217 94092959
Granulocyte-macrophage colony-stimulating factor (GM-CSF) reduces the density of stem cell factor receptors (c-kit oncogene product) on a GM-CSF-dependent human myeloid cell line.
Oez S; Hofmann-Wackersreuther G; Birkmann J; Smetak M; Welte K; Gallmeier WM
5. Medizinische Klinik, Klinikum der Stadt Nurnberg, Germany.
Eur Cytokine Netw (FRANCE) Jul-Aug 1993, 4 (4) p293-7, ISSN 1148-5493 Journal Code: A56
Languages: ENGLISH
Document type: JOURNAL ARTICLE
By employing a monoclonal *antibody* against the stem cell factor *receptor* (SCF-R), c-kit oncogene product, we analysed in flow cytometric technique the density of SCF-R on GM/SO cells which were incubated under various culture conditions. These experiments revealed that there is an inverse correlation between the SCF-R density on the cells and the doses of granulocyte-macrophage colony-stimulating factor (GM-CSF) in culture medium; the lower the dose, the higher the density of SCF-R on the cells. More detailed analyses showed that, in contrast to SCF which rapidly downregulates its own receptor, GM-CSF does not alter the measurable level of SCF-R in an exposition period of 60 minutes, which suggests that the internalization or shedding of the receptor is not the mechanism of action. Since the most striking difference regarding density of SCF-R between GM-CSF-treated and untreated cells was observed on day 2, the modulation of c-kit oncogene protein by GM-CSF likely occur prior to expression of protein onto the cell surface. In order to exclude the possibility that altered cell viability due to insufficient GM-CSF content in culture medium might be responsible for the increased SCF-R densities on GM-CSF-dependent cells, we subsequently generated a GM-CSF-independent subclone which still responded to GM-CSF as well as the dependent did. The experiments carried out with this subclone confirmed the results presented above. Thus our data suggest that GM-CSF is directly involved in the regulation of SCF receptor density on GM/SO cells.

10/7/36 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


07826533 94004056
Specific activation of human mast cells by the ligand for c-kit: comparison between lung, uterus and heart mast cells.
Sperr WR; Czerwenka K; Mundigler G; Muller MR; Semper H; Klappacher G; Glogar HD; Lechner K; Valent P
Department of Internal Medicine I, University of Vienna, Austria.
Int Arch Allergy Immunol (SWITZERLAND) 1993, 102 (2) p170-5, ISSN 1018-2438 Journal Code: BJ7
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Recent data suggest that stem cell factor (SCF or c-kit ligand, KL) is a

major regulator of human mast cells (MCs). In the present study, MCs derived from the lung (n = 8), uterus (n = 14) and heart (n = 4) were analyzed for expression of c-kit receptor and for responses to recombinant SCF. MCs of all organs tested were recognized by mAbs to c-kit (YB5.B8, SR-1) as assessed by combined toluidine blue/immunofluorescence staining. Activation by rhSCF (10 ng/ml, 60 min) resulted in histamine release from lung MCs (SCF 12.8 +/- 2.7% histamine release; control 2.8 +/- 0.8%, $p < 0.01$), uterus MCs (SCF 16.8 +/- 5.8%; control 5.2 +/- 2.5%, $p < 0.01$) and heart MCs (SCF 18.4 +/- 2.6%; control 1.7 +/- 0.23%, $p < 0.01$). Short-term pre-incubation with rhSCF (15 min) did not result in histamine secretion ($p > 0.05$), but in an increase (lung 2.4 +/- 1.0 fold; uterus 2.1 +/- 1.1 fold, and heart 2.0 +/- 0.4 fold) of alpha IgE-induced mediator release ($p < 0.05$). The effects of SCF were dose-dependent (maximum responses at 10-100 ng/ml) and dependent on extracellular calcium. A monoclonal antibody to SCF was found to inhibit the effects of SCF on MCs. Furthermore, MCs could be desensitized specifically by pre-incubation of MCs with rhSCF in Ca-free medium. Together, these data suggest that SCF triggers mediator secretion from MCs in various organs via binding to the c-kit receptor.

10/7/37 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


05317847 87275733
The gonadotropic function of insulin.
Poretsky L; Kalin MF
Endocr Rev (UNITED STATES) May 1987, 8 (2) p132-41, ISSN 0163-769X
Journal Code: EIK
Contract/Grant No.: HD-22738, HD, NICHD
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW
We have reviewed the role of insulin in ovarian physiology. Clinical observations and experimental data strongly support the hypothesis that insulin possesses gonadotropic activity, when acting alone or with FSH or LH. This idea is further supported by the recent discovery of insulin in follicular fluid. The idea that insulin has gonadotropic function can explain a variety of clinical observations, which otherwise are difficult to understand. For example, manifestations of ovarian hypofunction (primary amenorrhea, late menarche, anovulation, low pregnancy rate, and early menopause) in IDDM can be understood if it is accepted that insulin is necessary for the ovary to reach its full steroidogenic potential. The idea that insulin affects ovarian steroidogenesis also helps to understand the observation that hyperandrogenism frequently accompanies each of the various insulin-resistant states, regardless of the latter's etiology (e.g. genetic deficiency in the number of insulin *receptors*, antiinsulin *receptor* *antibodies*, *obesity* , etc.). The explanation for this association is based on the idea that hyperinsulinemia intensifies ovarian steroidogenesis, which manifests clinically as hyperandrogenism. Continuous stimulation of the ovary by insulin over a long period of time possibly produces morphological ovarian changes, such as hyperthecosis or polycystic changes; these changes commonly are observed among women with insulin resistance. The effects of insulin on ovarian cells are mediated possibly through binding of the peptide to its own receptor or to the IGF-1 receptor (the specificity spillover phenomenon). The latter could be an important mechanism in cases of insulin resistance. Potential mechanisms underlying the gonadotropic activity of insulin include direct effects on steroidogenic enzymes, modulation of FSH or LH receptor number, synergism with FSH or LH, or nonspecific enhancement of cell viability. The gonadotropic function of insulin adds yet another note to what has been termed a symphony of insulin action. Further investigation into this new area may yield greater insights not only into normal ovarian physiology, but also into the pathogeneses of such diverse entities as PCO, obesity, diabetes mellitus, and the syndromes of insulin resistance and acanthosis nigricans. (90 Refs.)

10/7/38 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


03849051 84158070
Insulin receptors and syndromes of insulin resistance.
Kahn CR
Diabetes Care (UNITED STATES) May-Jun 1982, 5 Suppl 1 p98-101, ISSN 0149-5992 Journal Code: EAG
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Whenever normal concentrations of insulin produce a biologic response that is less than normal, insulin resistance should be suspected. Insulin resistance may occur in many different disease states and may involve an entire organism, a single tissue or cell, or even a single metabolic pathway of insulin action. This report outlines current knowledge about the mechanism of insulin resistance, as well as specific examples of disease states in which it occurs.

10/7/39 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


03811745 82042077
[Regulation of insulin receptor expression and its place in the mechanism of insulin action]
Reguliatsiia ekspressii insulinovykh retseptorov i ikh mesto v mekhanizme deistviia insulina.
Bezdrobnyi IuV
Usp Sovrem Biol (USSR) Jul-Aug 1981, 92 (1) p35-48, ISSN 0042-1324 Journal Code: X6I
Languages: RUSSIAN
Document type: JOURNAL ARTICLE; REVIEW
(163 Refs.)

10/7/40 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


03777041 80187367
Role of insulin receptors in insulin-resistant states.
Kahn CR
Metabolism (UNITED STATES) May 1980, 29 (5) p455-66, ISSN 0026-0495 Journal Code: MUM
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The interaction of insulin with its receptor represents one of the key intermediate steps between secretion of insulin and its final biologic effects. Alterations in this interaction have been found in a number of disease states, including obesity, non-insulin-dependent diabetes mellitus (NIDDM), glucocorticoid excess, and acromegaly, as well as several rare forms of severe insulin resistance. The major factor regulating the receptor in obesity and NIDDM appears to be insulin. In obesity this alteration in normal regulation occurs secondary to overeating, whereas in the diabetic state the nature of the primary defect is uncertain. The role of the receptor in insulin resistance and methods for its evaluation are discussed.

10/7/41 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

03766255 82183231
Regulation of human blood erythroid burst-forming unit (BFU-E) proliferation by T-lymphocyte subpopulations defined by Fc *receptors* and *monoclonal* *antibodies*.
Mangan KF; Chikkappa G; Bieler LZ; Scharfman WB; Parkinson DR
Blood (UNITED STATES) May 1982, 59 (5) p990-6, ISSN 0006-4971
Journal Code: A8G
Contract/Grant No.: SO7-RR05394, RR, NCRR
Languages: ENGLISH
Document type: JOURNAL ARTICLE

10/7/42 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)


03096047 77259005
[New data in the domain of insulin resistance]
Donnees nouvelles dans le domaine de l'insulino-resistance.
Freychet P
Sem Hop (FRANCE) Jun 23 1977, 53 (24) p1421-4, ISSN 0037-1777
Journal Code: ULD
Languages: FRENCH Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE English Abstract
Insulin resistance may occur to a variable degree in various disease conditions. Obesity is frequently accompanied by insulin resistance. The anti-insulin antibodies in patients treated with insulin are a classical cause, but in fact rare. Insulin resistance of variable degree may accompany certain metabolic disorders, e.g. diabetic ketosis and acidosis, and endocrine disorders, e.g. Cushing's syndrome, acromegaly. The measurement of insulin receptors brings a new dimension to the investigation of insulin resistance. Insulin receptors are reduced in number during obesity. The abnormality, partly responsible for insulin resistance, is reducible by reduction in calory intake. Circulating insulin anti-*receptor* *antibodies* appear to be responsible for insulin resistance which is particularly marked although exeptional, in nonobese diabetics with acanthosis nigrans and auto-immune symptoms.

10/7/43 (Item 1 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


15804547 Genuine Article#: XH428 Number of References: 80
Title: Features of macrophage differentiation induced by p19(INK4d), a specific inhibitor of cyclin D-dependent kinases
Author(s): Adachi M; Roussel MF; Havenith K; Sheer CJ (REPRINT)
Corporate Source: ST JUDE CHILDRENS HOSP,DEPT TUMOR CELL BIOL, 332 N LAUDERDALE/MEMPHIS//TN/38105 (REPRINT); ST JUDE CHILDRENS HOSP,DEPT TUMOR CELL BIOL/MEMPHIS//TN/38105; ST JUDE CHILDRENS HOSP,DEPT IMMUNOL/MEMPHIS//TN/38105; ST JUDE CHILDRENS HOSP,HOWARD HUGHES MED INST/MEMPHIS//TN/38105
Journal: BLOOD, 1997, V90, N1 (JUL 1), P126-137
ISSN: 0006-4971 Publication date: 19970701
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: ARTICLE
Abstract: The mitogen-dependent induction of cyclin D-dependent kinase activity is required for cells to enter the DNA synthetic (S) phase of their division cycle. Immature 32Dcl3 myeloid cells (32D) proliferating in the presence of interleukin-3 (IL-3) normally express cyclins D2 and D3, which assemble into binary holoenzyme complexes with their catalytic subunits, CDK4 and CDK6. When 32D cells are switched to medium containing granulocyte colony-stimulating factor (G-CSF) instead of IL-3, D-type cyclins are degraded and, in the absence of their associated kinase activity, the cells arrest in the first gap phase

(G(1)) of the cell cycle and differentiate to neutrophils. We derived 32D cells in which the expression of p19(INK4d), a specific polypeptide inhibitor of CDK4 and CDK6, is regulated by the heavy metal-inducible sheep metallothionein promoter, Induction of p19(INK4d) in response to zinc prolonged cell survival in the absence of growth factor treatment, When maintained in medium containing both IL-3 and zinc, these cells lost cyclin D-dependent kinase activity, underwent G(1) phase arrest, and acquired certain morphologic, antigenic, and functional properties of mononuclear phagocytes, Cells induced to express p19(INK4d) did not synthesize receptors for macrophage colony-stimulating factor (M-CSF/CSF-1) and reverted to an immature myeloid phenotype when shifted back into medium containing IL-3 alone, These cells exhibited accelerated differentiation to neutrophils in response to G-CSF but also gave rise to macrophage-like cells when maintained in medium containing both G-CSF and zinc, Therefore, the acquisition of macrophage properties in response to zinc treatment neither depended upon IL-3 nor upon G(1) phase arrest per se and instead reflects some ability of p19(INK4d), and presumably cyclin D-dependent kinases, to affect myeloid differentiation, (C) 1997 by The American Society of Hematology.

10/7/44 (Item 2 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


15445995 Genuine Article#: WH236 Number of References: 52
Title: Cytoplasmic domains of the common beta-chain of the GM-CSF/IL-3/IL-5 receptors that are required for inducing differentiation or clonal suppression in myeloid leukaemic cell lines
Author(s): Smith A; Metcalf D; Nicola NA (REPRINT)
Corporate Source: WALTER & ELIZA HALL INST MED RES,PO ROYAL MELBOURNE HOSP/MELBOURNE/VIC 3050/AUSTRALIA/ (REPRINT); WALTER & ELIZA HALL INST MED RES,/MELBOURNE/VIC 3050/AUSTRALIA/; COOPERAT RES CTR CELLULAR GROWTH FACTORS,/MELBOURNE/VIC 3050/AUSTRALIA/
Journal: EMBO JOURNAL, 1997, V16, N3 (FEB 3), P451-464
ISSN: 0261-4189 Publication date: 19970203
Publisher: OXFORD UNIV PRESS UNITED KINGDOM, WALTON ST JOURNALS DEPT, OXFORD, ENGLAND OX2 6DP
Language: English Document Type: ARTICLE
Abstract: Granulocyte-macrophage colony stimulating factor (GM-CSF) is a cytokine that controls the production and function of myeloid cells by interaction with a cell surface receptor composed of a specific ligand-binding alpha-chain (hGMR alpha) and a shared signal-transducing beta-chain (beta c) Co-expression of human GMR alpha-chain and wild-type human beta c in two murine leukaemic cell lines (M1 and WEHI-3B D+) conferred the ability to terminally differentiate into macrophages when stimulated with human GM-CSF, Analysis of cytoplasmic truncation mutants of beta c showed that residues to amino acid 783 (numbering from the first amino acid of the leader sequence) were sufficient for the GM-CSF-dependent induction of all aspects of differentiation in both cell types. However, shorter truncations selectively lost, in a cell-specific manner, first the capacity to induce macrophage migration in agar and then cell surface differentiation antigens and clonal suppression of proliferative potential. The data suggest that different aspects of the differentiated phenotype can be dissociated with the required signalling pathways originating from distinct regions of the receptor cytoplasmic domain and cooperating to produce a fully differentiated macrophage. The cooperativity of these pathways and limiting cell signalling intermediate pool sizes could explain the observed cell line differences and may have implications for normal haemopoiesis.

10/7/45 (Item 3 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci

15381519 Genuine Article#: WD034 Number of References: 53
Title: Interactions between c-kit and stem cell factor are not required for B-cell development in vivo
Author(s): Takeda S (REPRINT) ; Shimizu T; Rodewald HR
Corporate Source: KYOTO UNIV,SCH MED, BAYER CHAIR DEPT MOL IMMUNOL, SAKYO KU/KYOTO 606//JAPAN/ (REPRINT); BASEL INST IMMUNOL,/BASEL//SWITZERLAND/
Journal: BLOOD, 1997, V89, N2 (JAN 15), P518-525
ISSN: 0006-4971 Publication date: 19970115
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: ARTICLE
Abstract: The receptor-type tyrosine kinase, c-kit is expressed in hematopoietic stem cells (HSC), myeloid, and lymphoid precursors. In c-kit ligand-deficient mice, absolute numbers of HSC are mildly reduced suggesting that c-kit is not essential for HSC development However, c-kit(-) HSC cannot form spleen colonies or reconstitute hematopoietic functions in lethally irradiated recipient mice. Based on in in vitro experiments, a critical role of c-kit in B-cell development was suggested. Here we have investigated the B-cell development of c-kit-null mutant (W/W) mice in vivo. Furthermore, day 13 fetal liver cells from wild type or W/W mice were transferred into immunodeficientRAG-2(-/-) mice. Surprisingly, transferred c-kit(-) cells gave rise to allstages of immature B cells in the bone marrow and subsequently to mature conventional B2, as well as B1, type B cells in the recipients to the same extent as transferred wild type cells. Hence, in contrast to important rolesof c-kit in the expansion of HSC and the generation of erythroid and myeloid lineages and T-cell precursors, c-kit- HSC can colonize the recipient bone marrow and differentiate into B cells in the absence of c-kit. (C) 1997 by The American Society of Hematology.

10/3/46 (Item 4 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


15076778 Genuine Article#: VH768 No. References: 50
Title: DEREGULATED EXPRESSION OF C-MYC IN MEGAKARYOCYTES OF TRANSGENIC MICE INCREASES MEGAKARYOPOIESIS AND DECREASES POLYPLOIDIZATION
Author(s): THOMPSON A; ZHANG Y; KAMEN D; JACKSON CW; CARDIFF RD; RAVID K
Corporate Source: BOSTON UNIV,SCH MED,DEPT BIOCHEM/BOSTON//MA/02139; BOSTON UNIV,SCH MED,DEPT BIOCHEM/BOSTON//MA/02139; ST JUDE CHILDRENS HOSP,DIV EXPT HEMATOL/MEMPHIS//TN/38105; UNIV CALIF DAVIS,DEPT MED PATHOL/SACRAMENTO//CA/95817
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1996, V271, N38 (SEP 20), P 22976-22982
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/47 (Item 5 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14631646 Genuine Article#: UA772 No. References: 52
Title: INCREASED EXPRESSION OF C-KIT OR ITS LIGAND STEEL FACTOR IS NOT A COMMON FEATURE OF ADULT ACUTE MYELOID-LEUKEMIA
Author(s): COLE SR; AYLETT GW; CASEY G; HARVEY NL; CAMBARERI AC; ASHMAN LK
Corporate Source: INST MED & VET SCI,HANSON CTR CANC RES,LEUKAEMIA RES UNIT,POB 14,RUNDLE MALL/ADELAIDE/SA 5000/AUSTRALIA/; INST MED & VET SCI,HANSON CTR CANC RES,LEUKAEMIA RES UNIT/ADELAIDE/SA 5000/AUSTRALIA/
Journal: LEUKEMIA, 1996, V10, N2 (FEB), P288-296
ISSN: 0887-6924
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/48 (Item 6 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14580474 Genuine Article#: TZ281 No. References: 64
Title: ACCELERATED IMMUNE-RESPONSE IN TRANSGENIC MICE EXPRESSING RAT CD44V4-V7 ON T-CELLS
Author(s): MOLL J; SCHMIDT A; VANDERPUTTEN H; PLUG R; PONTA H; HERRLICH P; ZOLLER M
Corporate Source: GERMAN CANC RES CTR,DEPT TUMOR PROGRESS & IMMUNE DEF,NEUENHEIMER FELD 280/D-69120 HEIDELBERG//GERMANY/; GERMAN CANC RES CTR,DEPT TUMOR PROGRESS & IMMUNE DEF/D-69120 HEIDELBERG//GERMANY/; NUCL RES CTR,INST GENET/KARLSRUHE//GERMANY/; CIBA GEIGY AG,CENT NERVOUS SYST,DEPT MOLEC & CELLULAR BIOL/BASEL//SWITZERLAND/
Journal: JOURNAL OF IMMUNOLOGY, 1996, V156, N6 (MAR 15), P2085-2094
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/49 (Item 7 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14480568 Genuine Article#: TQ509 No. References: 101
Title: DIFFERENTIAL EXPRESSION OF GRANULOCYTE-MACROPHAGE COLONY-STIMULATING FACTOR AND IL-3 RECEPTOR SUBUNITS ON HUMAN CD34(+) CELLS AND LEUKEMIC-CELL LINES
Author(s): KURATA H; ARAI T; YOKOTA T; ARAI K
Corporate Source: UNIV TOKYO,INST MED SCI,DEPT MOLEC & DEV BIOL,MINATO KU,4-6-1 SHIROKANEDAI/TOKYO 108//JAPAN/; UNIV TOKYO,INST MED SCI,DEPT MOLEC & DEV BIOL,MINATO KU/TOKYO 108//JAPAN/; SAITAMA MED SCH,GEN MED CTR,DEPT LAB MED/MOROYAMA/SAITAMA/JAPAN/; SCI UNIV TOKYO/TOKYO 162//JAPAN/
Journal: JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, 1995, V96, N6 (DEC), P 1083-1099
ISSN: 0091-6749
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/50 (Item 8 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14430351 Genuine Article#: TM195 No. References: 107
Title: HEMATOPOIESIS IN THE THYMUS
Author(s): KENDALL MD
Corporate Source: BABRAHAM INST,THYMUS LAB/CAMBRIDGE CB2 4AT//ENGLAND/; UNITED MED & DENT SCH GUYS & ST THOMASS HOSP,DEPT PHARMACOL/LONDON SE1 7EH//ENGLAND/
Journal: DEVELOPMENTAL IMMUNOLOGY, 1995, V4, N3, P157&
ISSN: 1044-6672
Language: ENGLISH Document Type: REVIEW (Abstract Available)

10/3/51 (Item 9 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14348389 Genuine Article#: TF891 No. References: 38
Title: IDENTIFICATION OF A UNIQUE MEMBRANE-BOUND MOLECULE ON A HEMATOPOIETIC STEM-CELL LINE AND ON MULTIPOTENT PROGENITOR CELLS
Author(s): HAN XD; CHUNG SW; WONG PMC
Corporate Source: TEMPLE UNIV,FELS INST CANC RES & MOLEC BIOL,DEPT LAB MED & PATHOL,3307 N BROAD ST/PHILADELPHIA//PA/19140; TEMPLE UNIV,FELS INST CANC RES & MOLEC BIOL,DEPT LAB MED & PATHOL/PHILADELPHIA//PA/19140;

TEMPLE UNIV,FELS INST CANC RES & MOLEC BIOL,DEPT BIOCHEM/PHILADELPHIA//PA/19140; TEMPLE UNIV,FELS INST CANC RES & MOLEC BIOL,DEPT MICROBIOL & IMMUNOL/PHILADELPHIA//PA/19140
Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 1995, V92, N24 (NOV 21), P11014-11018
ISSN: 0027-8424
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/52 (Item 10 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14137392 Genuine Article#: RU050 No. References: 46
Title: T-CELL PRECURSORS IN THE SPLEEN GIVE RISE TO COMPLEX T-CELL REPERTOIRES IN THE THYMUS AND THE INTESTINE
Author(s): HAMAD M; WHETSELL M; KLEIN JR
Corporate Source: UNIV TULSA,DEPT BIOL SCI,600 S COLL AVE/TULSA//OK/74104; UNIV TULSA,DEPT BIOL SCI/TULSA//OK/74104; UNIV TULSA,MEERVIN BOVAIRD CTR STUDIES MOLEC BIOL & BIOTECHN/TULSA//OK/74104
Journal: JOURNAL OF IMMUNOLOGY, 1995, V155, N6 (SEP 15), P2866-2876
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/53 (Item 11 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


14127222 Genuine Article#: RT386 No. References: 69
Title: THROMBOCYTOPENIA IN DOGS INDUCED BY GRANULOCYTE-MACROPHAGE COLONY-STIMULATING FACTOR - INCREASED DESTRUCTION OF CIRCULATING PLATELETS
Author(s): NASH RA; BURSTEIN SA; STORB R; YANG WJ; ABRAMS K; APPELBAUM FR; BOONE T; DEEG HJ; DURACK LD; SCHUENING FG; MCDONOUGH S; MOORE P; NELP WB; SLICHTER S
Corporate Source: UNIV WASHINGTON,SCH MED,FRED HUTCHINSON CANC RES CTR,1124 COLUMBIA ST,M318/SEATTLE//WA/98104; PUGET SOUND BLOOD CTR/SEATTLE//WA/98104; UNIV OKLAHOMA/NORMAN//OK/73019; UNIV CALIF DAVIS/DAVIS//CA/95616; AMGEN INC/THOUSAND OAKS//CA/91320
Journal: BLOOD, 1995, V86, N5 (SEP 1), P1765-1775
ISSN: 0006-4971
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/54 (Item 12 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


13910359 Genuine Article#: QZ139 No. References: 31
Title: ANALYSIS OF C-KIT EXPRESSION OF HUMAN ERYTHROLEUKEMIA CELL-LINE, HEL - CLONAL VARIATION AND RELATIONSHIP WITH ERYTHROID AND MEGAKARYOCYTIC PHENOTYPE
Author(s): KUBOTA A; OKAMURA S; SHIMODA K; IKEMATSU W; OTSUKA T; NIHO Y
Corporate Source: KYUSHU UNIV,FAC MED,DEPT INTERNAL MED 1,HIGASHIKU,3-1-1 MAIDASHI/FUKUOKA 812//JAPAN/; KYUSHU UNIV,FAC MED,CTR CANC,HIGASHI KU/FUKUOKA812//JAPAN/
Journal: LEUKEMIA RESEARCH, 1995, V19, N4 (APR), P283-290
ISSN: 0145-2126
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/55 (Item 13 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci

13901077 Genuine Article#: RA031 No. References: 66
Title: GATA-1 REPROGRAMS AVIAN MYELOMONOCYTIC CELL-LINES INTO EOSINOPHILS, THROMBOBLASTS, AND ERYTHROBLASTS
Author(s): KULESSA H; FRAMPTON J; GRAF T
Corporate Source: EUROPEAN MOLEC BIOL LAB,DIFFERENTIAT PROGRAMME/D-69117 HEIDELBERG//GERMANY/
Journal: GENES & DEVELOPMENT, 1995, V9, N10 (MAY 15), P1250-1262
ISSN: 0890-9369
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/56 (Item 14 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

13886833 Genuine Article#: QY218 No. References: 38
Title: ENHANCED EXPRESSION OF CD34 MESSENGER-RNA BY DEVELOPING ENDOTHELIAL-CELLS OF MICE
Author(s): ITO A; NOMURA S; HIROTA S; SUDA J; SUDA T; KITAMURA Y
Corporate Source: OSAKA UNIV,SCH MED,DEPT PATHOL,YAMADA OKA 2-2/SUITA/OSAKA 565/JAPAN/; OSAKA UNIV,SCH MED,DEPT PATHOL/SUITA/OSAKA 565/JAPAN/; KUMAMOTO UNIV,SCH MED,IMEG,DEPT CELL DIFFERENTIAT/KUMAMOTO 860//JAPAN/
Journal: LABORATORY INVESTIGATION, 1995, V72, N5 (MAY), P532-538
ISSN: 0023-6837
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/57 (Item 15 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

13870422 Genuine Article#: QX865 No. References: 38
Title: ADHESIVE INTERACTIONS BETWEEN ALTERNATIVELY SPLICED CD44 ISOFORMS
Author(s): DROLL A; DOUGHERTY ST; CHIU RK; DIRKS JF; MCBRIDE WH; COOPER DL; DOUGHERTY GJ
Corporate Source: BRITISH COLUMBIA CANC AGCY,TERRY FOX LAB,TUMOURIMMUNOL GRP,601 W 10TH AVE/VANCOUVER/BC V5Z 1L3/CANADA/; BRITISH COLUMBIA CANC AGCY,DEPT RADIAT ONCOL/VANCOUVER/BC V5Z 1L3/CANADA/; UNIV CALIF LOS ANGELES,MED CTR,DEPT RADIAT ONCOL/LOS ANGELES//CA/90024; UNIV PITTSBURGH,SCH MED,DEPT PATHOL/PITTSBURGH//PA/15261
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1995, V270, N19 (MAY 12), P 11567-11573
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/58 (Item 16 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

13304846 Genuine Article#: PF189 No. References: 78
Title: MONOCYTE ADHESION IN PATIENTS WITH BONE-MARROW FIBROSIS IS REQUIRED FOR THE PRODUCTION OF FIBROGENIC CYTOKINES - POTENTIAL ROLE FOR INTERLEUKIN-1 AND TGF-BETA
Author(s): RAMESHWAR P; DENNY TN; STEIN D; GASCON P
Corporate Source: UNIV MED & DENT NEW JERSEY,NEW JERSEY MED SCH,DEPT MED,DIV HEMATOL,MSB,ROOM E585/NEWARK//NJ/07103; UNIV MED & DENT NEW JERSEY,NEW JERSEY MED SCH,DEPT MED,DIV HEMATOL/NEWARK//NJ/07103; UNIV MED & DENT NEW JERSEY,NEW JERSEY MED SCH,DEPT PEDIAT/NEWARK//NJ/07103
Journal: JOURNAL OF IMMUNOLOGY, 1994, V153, N6 (SEP 15), P2819-2830
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/59 (Item 17 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci

13283021 Genuine Article#: PC990 No. References: 35
Title: MURINE ERYTHROLEUKEMIA (MEL) CELLS BEAR LIGANDS FOR THE SIALOADHESIN AND ERYTHROBLAST RECEPTOR MACROPHAGE HEMAGGLUTININS
Author(s): FRASER IP; GORDON S
Corporate Source: UNIV OXFORD,SIR WILLIAM DUNN SCH PATHOL,S PARKSRD/OXFORD OX1 3RE//ENGLAND/
Journal: EUROPEAN JOURNAL OF CELL BIOLOGY, 1994, V64, N2 (AUG), P217-221
ISSN: 0171-9335
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/60 (Item 18 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


13166513 Genuine Article#: NT761 No. References: 41
Title: UTILIZATION OF THE BETA-CHAIN AND GAMMA-CHAIN OF THE IL-2 RECEPTOR BY THE NOVEL CYTOKINE-IL-15
Author(s): GIRI JG; AHDIEH M; EISENMAN J; SHANEBECK K; GRABSTEIN K; KUMAKI S; NAMEN A; PARK LS; COSMAN D; ANDERSON D
Corporate Source: IMMUNEX RES & DEV CORP,51 UNIV ST/SEATTLE//WA/98101
Journal: EMBO JOURNAL, 1994, V13, N12 (JUN 15), P2822-2830
ISSN: 0261-4189
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/61 (Item 19 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


13148998 Genuine Article#: NR989 No. References: 24
Title: CLASS-I DEPENDENCE OF THE DEVELOPMENT OF CD4+CD8-NK1.1+ THYMOCYTES
Author(s): COLES MC; RAULET DH
Corporate Source: UNIV CALIF BERKELEY,DEPT MOLEC & CELL BIOL,DIV IMMUNOL,489 LSA/BERKELEY//CA/94720; UNIV CALIF BERKELEY,DEPT MOLEC & CELL BIOL,DIV IMMUNOL/BERKELEY//CA/94720; UNIV CALIF BERKELEY,CANC RES LAB/BERKELEY//CA/94720
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1994, V180, N1 (JUL 1), P395-399
ISSN: 0022-1007
Language: ENGLISH Document Type: NOTE (Abstract Available)

10/3/62 (Item 20 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


13064974 Genuine Article#: NM615 No. References: 70
Title: CONSERVED AMINO-ACID-RESIDUES IN THE COMPLEMENTARITY-DETERMINING REGION-1 OF THE TCR BETA-CHAIN ARE INVOLVED IN THE RECOGNITION OF CONVENTIONAL AG AND MLS-1 SUPERANTIGEN
Author(s): KANG JS; CHAMBERS CA; PAWLING J; SCOTT C; HOZUMI N
Corporate Source: UNIV TORONTO,MT SINAI HOSP,SAMUEL LUNENFELD RESINST,DIV MOLEC IMMUNOL & NEUROBIOL,600 UNIV AVE/TORONTO M5G 1X5/ON/CANADA/; UNIV TORONTO,MT SINAI HOSP,SAMUEL LUNENFELD RESINST,DIV MOLEC IMMUNOL & NEUROBIOL/TORONTO M5G 1X5/ON/CANADA/; UNIV TORONTO,DEPT MED GENET/TORONTO/ON/CANADA/; UNIV TORONTO,DEPT IMMUNOL/TORONTO/ON/CANADA/
Journal: JOURNAL OF IMMUNOLOGY, 1994, V152, N11 (JUN 1), P5305-5317
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/63 (Item 21 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci

12714487 Genuine Article#: MJ284 No. References: 18
Title: EXPRESSION OF ANTIGENS ON HEMATOPOIETIC PROGENITOR CELLS IN BOVINE BONE-MARROW
Author(s): MUIYA P; LOGANHENFREY L; NAESSENS J
Corporate Source: INT LAB RES ANIM DIS,POB 30709/NAIROBI//KENYA/; INT LAB RES ANIM DIS,POB 30709/NAIROBI//KENYA/
Journal: VETERINARY IMMUNOLOGY AND IMMUNOPATHOLOGY, 1993, V39, N1-3 (NOV) , P237-248
ISSN: 0165-2427
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/64 (Item 22 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

12493797 Genuine Article#: LR771 No. References: 28
Title: POSITIVE SELECTION OF T-LYMPHOCYTES ON FIBROBLASTS
Author(s): PAWLOWSKI T; ELLIOTT JD; LOH DY; STAERZ UD
Corporate Source: NATL JEWISH CTR IMMUNOL & RESP MED,DEPT MED,1400 JACKSON ST/DENVER//CO/80206; WASHINGTON UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT MED/ST LOUIS//MO/63110; WASHINGTON UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT GENET/ST LOUIS//MO/63110; WASHINGTON UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT MICROBIOL/ST LOUIS//MO/63110; WASHINGTON UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT IMMUNOL/ST LOUIS//MO/63110; UNIV COLORADO,HLTH SCI CTR,DEPT MICROBIOL IMMUNOL/DENVER//CO/80262; UNIV COLORADO,HLTH SCI CTR,CTR CANC/DENVER//CO/80262
Journal: NATURE, 1993, V364, N6438 (AUG 12), P642-645
ISSN: 0028-0836
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/65 (Item 23 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

12470367 Genuine Article#: LP362 No. References: 41
Title: LOW-AFFINITY RECEPTORS FOR TUMOR-NECROSIS-FACTOR-ALPHA, INTERFERON-GAMMA AND GRANULOCYTE-MACROPHAGE COLONY-STIMULATING FACTOR ARE EXPRESSED ON HUMAN PLACENTAL SYNCYTIOTROPHOBLAST
Author(s): HAMPSON J; MCLAUGHLIN PJ; JOHNSON PM
Corporate Source: UNIV LIVERPOOL,DEPT IMMUNOL,POB 147/LIVERPOOL L69 3BX//ENGLAND/; UNIV LIVERPOOL,DEPT IMMUNOL,POB 147/LIVERPOOL L69 3BX//ENGLAND/; UNIV LIVERPOOL,DEPT HAEMATOL/LIVERPOOL L69 3BX//ENGLAND/
Journal: IMMUNOLOGY, 1993, V79, N3 (JUL), P485-490
ISSN: 0019-2805
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/66 (Item 24 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

12456515 Genuine Article#: LN625 No. References: 48
Title: SELF-RENEWAL AND DIFFERENTIATION OF NORMAL AVIAN ERYTHROID PROGENITOR CELLS - REGULATORY ROLES OF THE TGF-ALPHA/C-ERBB AND SCF/C-KIT RECEPTORS
Author(s): HAYMAN MJ; MEYER S; MARTIN F; STEINLEIN P; BEUG H
Corporate Source: SUNY,DEPT MICROBIOL/STONY BROOK//NY/11794; AMGEN CTR/THOUSAND OAKS//CA/91320; INST MOLEC PATHOL/A-1030 VIENNA//AUSTRIA/
Journal: CELL, 1993, V74, N1 (JUL 16), P157-169
ISSN: 0092-8674
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/67 (Item 25 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


12268209 Genuine Article#: KY452 No. References: 68
Title: IFN-GAMMA INFLUENCES THE MIGRATION OF THORACIC-DUCT B-LYMPHOCYTE AND T-LYMPHOCYTE SUBSETS INVIVO - RANDOM INCREASE IN DISAPPEARANCE FROM THE BLOOD AND DIFFERENTIAL DECREASE IN REAPPEARANCE IN THE LYMPH
Author(s): WESTERMANN J; PERSIN S; MATYAS J; VANDERMEIDE P; PABST R
Corporate Source: MED SCH HANNOVER,CTR ANAT,ABT 2,4120,POSTFACH 610180/W-3000 HANNOVER 61//GERMANY/; TNO,INST RADIOBIOL & APPL IMMUNOL/RIJSWIJK//NETHERLANDS/
Journal: JOURNAL OF IMMUNOLOGY, 1993, V150, N9 (MAY 1), P3843-3852
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/68 (Item 26 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


12214258 Genuine Article#: KV696 No. References: 60
Title: BONE-MARROW STROMA IN HUMANS - ANTI-NERVE GROWTH-FACTOR *RECEPTOR* *ANTIBODIES* SELECTIVELY STAIN RETICULAR CELLS INVIVO AND INVITRO
Author(s): CATTORETTI G; SCHIRO R; ORAZI A; SOLIGO D; COLOMBO MP
Corporate Source: IST NAZL STUDIO & CURA TUMORI,DIV ANAT PATHOL &CYTOL,VIA VENEZIAN 1/I-20133 MILAN//ITALY/; IST NAZL STUDIO & CURA TUMORI,DIV ONCOL SPERIMENTALE D/MILAN//ITALY/; OSPED S GERARDO TINTORI/MONZA//ITALY/; UNIV MILAN,PEDIAT CLIN/I-20122 MILAN//ITALY/; INDIANA UNIV,SCH MED,DEPT PATHOL/INDIANAPOLIS//IN/46202; INDIANA UNIV,SCH MED,DIV HEMATOPATHOL/INDIANAPOLIS//IN/46202; UNIV MILAN,IST SCI MED/I-20122 MILAN//ITALY/
Journal: BLOOD, 1993, V81, N7 (APR 1), P1726-1738
ISSN: 0006-4971
Language: ENGLISH Document Type: ARTICLE

10/3/69 (Item 27 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


11916004 Genuine Article#: JW798 No. References: 33
Title: INTERLEUKIN-4 REGULATES INDUCTION OF SIALOADHESIN, THE MACROPHAGE SIALIC ACID-SPECIFIC RECEPTOR
Author(s): MCWILLIAM AS; TREE P; GORDON S
Corporate Source: UNIV OXFORD,SIR WILLIAM DUNN SCH PATHOL/OXFORD OX1 3RE//ENGLAND/; UNIV OXFORD,SIR WILLIAM DUNN SCH PATHOL/OXFORD OX1 3RE//ENGLAND/
Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 1992, V89, N21 (NOV 1), P10522-10526
ISSN: 0027-8424
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/70 (Item 28 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


11046591 Genuine Article#: GB983 No. References: 43
Title: PHENOTYPIC ANALYSIS OF SKIN INFILTRATES IN COMPARISON WITH PERIPHERAL-BLOOD LYMPHOCYTES, SPLEEN-CELLS AND THYMOCYTES IN EARLY AVIAN SCLERODERMA
Author(s): GRUSCHWITZ MS; MOORMANN S; KROMER G; SGONC R; DIETRICH H; BOECK G; GERSHWIN ME; BOYD R; WICK G
Corporate Source: UNIV INNSBRUCK,SCH MED,INST GEN & EXPTL PATHOL,FRITZ PREGL STR 3/A-6020 INNSBRUCK//AUSTRIA/; UNIV INNSBRUCK,SCH MED,INST GEN

& EXPTL PATHOL,FRITZ PREGL STR 3/A-6020 INNSBRUCK//AUSTRIA/; MONASH UNIV,DEPT PATHOL & IMMUNOL/PRAHRAN/VIC 3181/AUSTRALIA/; UNIV ERLANGEN NURNBERG/D-8520 ERLANGEN//FED REPGER/; UNIV CALIF DAVIS,SCH MED,DEPT INTERNAL MED,DIV RHEUMATOL CLIN IMMUNOL/DAVIS//CA/95616; UNIV CALIF DAVIS,SCH MED,DEPT AVIAN SCI/DAVIS//CA/95616
Journal: JOURNAL OF AUTOIMMUNITY, 1991, V4, N4, P577-593
Language: ENGLISH Document Type: ARTICLE

10/3/71 (Item 29 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

10945873 Genuine Article#: FU897 No. References: 46
Title: EXPRESSION AND FUNCTION OF C-KIT IN HEMATOPOIETIC PROGENITOR CELLS
Author(s): OGAWA M; MATSUZAKI Y; NISHIKAWA S; HAYASHI SI; KUNISADA T; SUDO T; KINA T; NAKAUCHI H; NISHIKAWA SI
Corporate Source: KUMAMOTO UNIV,SCH MED,INST MED IMMUNOL,DEPT PATHOL,2-2-1 HONJO/KUMAMOTO 860//JAPAN/; INST PHYS & CHEM RES FRONTIER RES PROGRAM,MOLECREGULAT AGING LAB/TSUKUBA/IBARAKI 305/JAPAN/; BIOMAT RES INST CO LTD/YOKOHAMA/KANAGAWA 244/JAPAN/; KYOTO UNIV,CHEST DIS RES INST,DEPT MOLEC PATHOL/KYOTO 606//JAPAN/
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1991, V174, N1, P63-71
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/72 (Item 30 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

10939927 Genuine Article#: FU768 No. References: 56
Title: EXPRESSION OF THE YB5.B8 ANTIGEN (C-KIT PROTOONCOGENE PRODUCT) IN NORMAL HUMAN BONE-MARROW
Author(s): ASHMAN LK; CAMBARERI AC; TO LB; LEVINSKY RJ; JUTTNER CA
Corporate Source: UNIV ADELAIDE,DEPT MICROBIOL & IMMUNOL,GPO BOX 498/ADELAIDE/SA 5001/AUSTRALIA/; INST MED & VET SCI,DIV HAEMATOL/ADELAIDE/SA 5000/AUSTRALIA/; INST CHILD HLTH,HUGH GREENWOOD DEPT IMMUNOL/LONDON WC1N 1EH//ENGLAND/
Journal: BLOOD, 1991, V78, N1, P30-37
Language: ENGLISH Document Type: NOTE

10/3/73 (Item 31 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

10780531 Genuine Article#: FF082 No. References: 34
Title: EXPRESSION OF HUMAN CSF-1 RECEPTOR INDUCES CSF-1-DEPENDENT PROLIFERATION IN MURINE MYELOID BUT NOT IN T-LYMPHOID CELLS
Author(s): VONRUDEN T; MOUCHIROUD G; BOURETTE RP; OUAZANA R; BLANCHET JP; WAGNER EF
Corporate Source: RES INST MOLEC PATHOL,DR BOHRGASSE 7/A-1030 VIENNA//AUSTRIA/; UNIV CLAUDE BERNARD LYON 1,CTR GENET MOLEC & CELLULAIRE,CNRS,UMR 106/VILLEURBANNE//FRANCE/
Journal: LEUKEMIA, 1991, V5, N1, P3-7
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/74 (Item 32 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci
(c) 1997 Inst for Sci Info. All rts. reserv.

10592710 Genuine Article#: EQ976 No. References: 59
Title: MEGALOBLASTIC HEMATOPOIESIS INVITRO - INTERACTION OF ANTIFOLATE *RECEPTOR* *ANTIBODIES* WITH *HEMATOPOIETIC* PROGENITOR CELLS LEADS TO A PROLIFERATIVE RESPONSE INDEPENDENT OF MEGALOBLASTIC CHANGES

Author(s): ANTONY AC; BRIDDELL RA; BRANDT JE; STRANEVA JE; VERMA RS; MILLER ME; KALASINSKI LA; HOFFMAN R
Corporate Source: INDIANA UNIV,SCH MED,DEPT MED,DIV HEMATOL ONCOL/INDIANAPOLIS//IN/46202
Journal: JOURNAL OF CLINICAL INVESTIGATION, 1991, V87, N1, P313-325
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

10/3/75 (Item 33 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


09741587 Genuine Article#: AT717 No. References: 183
Title: MONOCLONAL-ANTIBODIES IN CHARACTERIZATION OF NORMAL AND MALIGNANT HUMAN MYELOID CELLS
Author(s): ANDREASEN RB
Corporate Source: SPURVESKJULSPARKEN 4/DK-2830 VIRUM//DENMARK/; RIGSHOSP,DEPT MED A/COPENHAGEN//DENMARK/; PANUM INST,CANC BIOL LAB/COPENHAGEN//DENMARK/
Journal: DANISH MEDICAL BULLETIN, 1989, V36, N4, P303-316
Language: ENGLISH Document Type: REVIEW

10/3/76 (Item 34 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


09259680 Genuine Article#: R8173 No. References: 26
Title: EXPRESSION OF CD6 AND THE UCHL1-DEFINED CD45 (P180) ANTIGEN BY HUMAN COLONIC LYMPHOCYTES-T
Author(s): SMART CJ; HEATLEY RV; TREJDOSIEWICZ LK
Corporate Source: UNIV LEEDS,ST JAMES HOSP,DEPT MED/LEEDS LS9 7TF/W YORKSHIRE/ENGLAND/
Journal: IMMUNOLOGY, 1989, V66, N1, P90-95
Language: ENGLISH Document Type: ARTICLE

10/3/77 (Item 35 from file: 434)
DIALOG(R)File 434:Scisearch(R) Cited Ref Sci


05207171 Genuine Article#: QV863 No. References: 1
Title: I-123-INSULIN METABOLISM IN ANIMAL-MODELS OF INSULIN RESISTANCE SYNDROMES - EFFECT OF *ANTIBODIES* TO THE INSULIN-*RECEPTOR* AND *OBESITY*
Author(s): SODOYEZ JC; SODOYEZGOFFAUX F; TREVES S
Journal: DIABETES, 1983, V32, S1, PA43
Language: ENGLISH Document Type: MEETING ABSTRACT

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? show files
File 351:DERWENT WPI 1963-1997/UD=9736;UP=9733;UM=9730
         (c)1997 Derwent Info Ltd
File 348:EUROPEAN PATENTS 1978-1997/Sep W1
         (c) 1997 EUROPEAN PATENT OFFICE
File 376:Derwent Drug File 1964-1982
         (c) 1995 Derwent Info Ltd.
File 377:Derwent Drug File 1983-1997/Sep W4
         (c) 1997 Derwent Info Ltd.
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Set     Items   Description
S1        163   (LEPTIN? OR OB OR OBES? OR WSX OR HEMATOPOIET? OR HAEMATOP-
            OIET? OR HEMOPOIET? OR HAEMOPOIET?)(5N)RECEPTOR?
S2         45   S1 AND (ANTIBOD? OR ABS OR AB OR MAB? ? OR PAB? ? OR MONOC-
            LONAL)
S3         45   RD (unique items)
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? t s3/3/1-45

 3/3/1     (Item 1 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

011407446
WPI Acc No: 97-385353/199735
XRAM Acc No: C97-123627
XRPX Acc No: N97-320765
  Detecting defective *leptin* *receptor* by hybridisation assay - and
  treatment of obesity with agent that inhibits the defective receptor,
  also screening for compounds that supplement leptin activity
Patent Assignee: PROGENITOR INC (PROG-N)
Inventor: CIOFFI J; SHAFER A W; SNODGRASS H R; ZUPANCIC T J
Patent Family:
Patent No  Kind  Date   Applicat No Kind  Date   Main IPC      Week
WO 9726370  A1 19970724 WO 97US570   A  19970117               199735 B

Priority Applications (No Type Date): US 96588190 A 19960118
Filing Details:
Patent    Kind Filing Notes     Application  Patent
WO 9726370  A1
   Designated States (National): AL AM AU AZ BA BB BG BR BY CA CN CU CZ EE
   GE HU IL IS JP KG KP KR KZ LC LK LR LT LV MD MG MK MN MX NO NZ PL RO RU
   SG SI SK TJ TM TR TT UA UZ VN
   Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE
   LS LU MC MW NL OA PT SD SE SZ UG
Language, Pages: WO 9726370 (E, 26)


 3/3/2     (Item 2 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

011407431
WPI Acc No: 97-385338/199735
XRAM Acc No: C97-123612
XRPX Acc No: N97-320758
  *Leptin* *receptor*, *OB*-R, polypeptide - useful to treat obesity,
  optionally in conjuction with treatment for diabetes, high blood pressure
  and high cholesterol
Patent Assignee: UNIV ROCKEFELLER (UYRQ  )
Inventor: FRIEDMAN J M; IOFFE E; LEE G; PROENCA R
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Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9726335 A1 19970724 WO 97US1010 A 19970116 199735 B

Priority Applications (No Type Date): US 96599974 A 19960214; US 96586594 A 19960116
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9726335 A1
Designated States (National): AL AU BA BB BG BR CA CN CU CZ EE GE HU IL IS JP KP KR LC LK LR LT LV MG MK MN MW NO NZ PL RO SG SI SK TR TT UA UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
Language, Pages: WO 9726335 (E, 171)

3/3/3 (Item 3 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

011407384
WPI Acc No: 97-385291/199735
XRAM Acc No: C97-123565
XRPX Acc No: N97-320746
Detecting defective form of *leptin* *receptor* by probing cellular RNA - with oligonucleotide derived from DNA of *receptor* variant, also treatment of *obesity* by inhibiting expression of variant *receptor* and screening for agents that increase leptin activity
Patent Assignee: PROGENITOR INC (PROG-N)
Inventor: CIOFFI J; SHAFER A W; SNODGRASS H R; ZUPANCIC T J
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9726272 A1 19970724 WO 97US880 A 19970117 199735 B

Priority Applications (No Type Date): US 96588189 A 19960118
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9726272 A1
Designated States (National): AL AM AU AZ BA BB BG BR BY CA CN CU CZ EE GE HU IL IS JP KG KP KR KZ LC LK LR LT LV MD MG MK MN MX NO NZ PL RO RU SG SI SK TJ TM TR TT UA UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
Language, Pages: WO 9726272 (E, 26)

3/3/4 (Item 4 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

011394957
WPI Acc No: 97-372864/199734
XRAM Acc No: C97-120207
XRPX Acc No: N97-309574
*WSX* *receptor* and related *antibodies* and ligands - used to develop products for diagnosis and therapy, e.g. for improving haematopoiesis or for treating tumours
Patent Assignee: GENENTECH INC (GETH )
Inventor: BENNETT B; CARTER P J; CHIANG N Y; KIM K J; MATTHEWS W; RODRIGUES M L
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9725425 A1 19970717 WO 97US325 A 19970107 199734 B

Priority Applications (No Type Date): US 96667197 A 19960620; US 96585005 A

19960108
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9725425 A1
Designated States (National): AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
Language, Pages: WO 9725425 (E, 219)

3/3/5 (Item 5 from file: 351)
DIALOG(R)File 351:DERWENT WPI


011389153
WPI Acc No: 97-367060/199734
XRAM Acc No: C97-117736
*Monoclonal* *antibody* to human stem cell factor - comprises specifically binding to human stem cell factor, useful in diagnosis of blood diseases
Patent Assignee: NICHIREI KK (NCHK )
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
JP 9154578 A 19970617 JP 95335685 A 19951201 199734 B

Priority Applications (No Type Date): JP 95335685 A 19951201
Language, Pages: JP 9154578 (5)

3/3/6 (Item 6 from file: 351)
DIALOG(R)File 351:DERWENT WPI


011385716
WPI Acc No: 97-363623/199733
XRAM Acc No: C97-116561
Use of *monoclonal* *antibody* antagonists - for inhibiting interleukin-3 or other haemopoietic growth factors, for treating, e.g. leukaemia, lymphoma or allergy
Patent Assignee: MEDVET SCI PTY LTD (MEDV-N)
Inventor: LOPEZ A F
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9724373 A1 19970710 WO 96AU840 A 19961224 199733 B

Priority Applications (No Type Date): AU 967418 A 19960104; AU 957368 A 19951229
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9724373 A1
Designated States (National): AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
Language, Pages: WO 9724373 (E, 39)

3/3/7 (Item 7 from file: 351)
DIALOG(R)File 351:DERWENT WPI

011281114
WPI Acc No: 97-259018/199723
XRAM Acc No: C97-083737
XRPX Acc No: N97-214140
DNA encoding animal *haemopoietin* *receptor* which interacts with interleukin-13 - useful to treat asthma, allergy or condition exacerbated by IgE production
Patent Assignee: AMRAD OPERATIONS PTY LTD (AMRA-N)
Inventor: HILTON D J; METCALF D; NICOLA N A; WILLSON T; ZHANG J G
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9715663 | A1 | 19970501 | WO 96AU668 | A | 19961023 | | 199723 B |
| AU 9672668 | A | 19970515 | AU 9672668 | A | 19961023 | | 199736 |

Priority Applications (No Type Date): AU 962208 A 19960909; AU 956135 A 19951023; AU 957276 A 19951222
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9715663 | A1 | | | |

Designated States (National): AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG

| | | | | |
|---|---|---|---|---|
| AU 9672668 | A | Based on | | WO 9715663 |

Language, Pages: WO 9715663 (E, 90)

3/3/8 (Item 8 from file: 351)
DIALOG(R)File 351:DERWENT WPI


011234993
WPI Acc No: 97-212896/199719
XRAM Acc No: C97-068814
XRPX Acc No: N97-175550
Human *haemopoietin* *receptor* NR2, and corresponding DNA - used e.g. for treatment of auto-immune diseases
Patent Assignee: AMRAD OPERATIONS PTY LTD (AMRA-N)
Inventor: ALEXANDER W S; GAINSFORD T; HILTON D J; METCALF D; NG A; NICOLA N A; WILLSON T
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9712037 | A1 | 19970403 | WO 96AU607 | A | 19960926 | | 199719 B |
| AU 9669805 | A | 19970417 | AU 9669805 | A | 19960926 | | 199732 |

Priority Applications (No Type Date): AU 955641 A 19950926
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9712037 | A1 | | | |

Designated States (National): AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
Designated States (Regional): AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG

| | | | | |
|---|---|---|---|---|
| AU 9669805 | A | Based on | | WO 9712037 |

Language, Pages: WO 9712037 (E, 96)

3/3/9 (Item 9 from file: 351)
DIALOG(R)File 351:DERWENT WPI


011021725

WPI Acc No: 96-518675/199651
XRAM Acc No: C96-162913
XRPX Acc No: N96-437004
Nucleic acid encoding obesity protein - useful for treating obesity or associated diseases e.g. type II diabetes.
Patent Assignee: CHIRON CORP (CHIR )
Inventor: GIESE K W; WILLIAMS L T
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9635787 | A1 | 19961114 | WO 96US6609 | A | 19960508 | C12N-015/12 | 199651 B |
| AU 9657376 | A | 19961129 | AU 9657376 | A | 19960508 | C12N-015/12 | 199712 |

Priority Applications (No Type Date): US 95437834 A 19950508
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9635787 | A1 | | | |
| | | Designated States (National): AU CA CN JP MX | | |
| | | Designated States (Regional): AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE | | |
| AU 9657376 | A | Based on | | WO 9635787 |

Language, Pages: WO 9635787 (E, 66)

3/3/10 (Item 10 from file: 351)
DIALOG(R)File 351:DERWENT WPI


011000683 **Image available**
WPI Acc No: 96-497632/199649
XRAM Acc No: C96-155614
XRPX Acc No: N96-419599
Immortalised pre-adipocytes contg viral oncogene fragment - useful for identifying cpds that regulate lipolysis and thermogenesis, as lipolytic agents and models for studying adipocyte processes
Patent Assignee: CNRS CENT NAT RECH SCI (CNRS )
Inventor: STROSBERG A D; ZILBERFARB V
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9634100 | A1 | 19961031 | WO 96FR634 | A | 19960425 | C12N-015/12 | 199649 B |
| FR 2733513 | A1 | 19961031 | FR 954922 | A | 19950425 | C12N-005/06 | 199701 |

Priority Applications (No Type Date): FR 954922 A 19950425
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9634100 | A1 | | | |
| | | Designated States (National): CA JP US | | |
| | | Designated States (Regional): AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE | | |

Language, Pages: WO 9634100 (F, 52); FR 2733513 (30)

3/3/11 (Item 11 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010969759
WPI Acc No: 96-466708/199647
XRAM Acc No: C96-146491
XRPX Acc No: N96-393081
New *monoclonal* *antibodies* against gp130 - mimicking effector actions of some cytokine(s), useful therapeutically, as cell culture adjuvant or as diagnostic reagent
Patent Assignee: DIACLONE SA (DIAC-N)
Inventor: CLEMENT C; WIJDENES J
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|

EP 738736 A1 19961023 EP 96400799 A 19960415 C07K-016/28 199647 B
FR 2733250 A1 19961025 FR 954809 A 19950421 C12P-021/08 199649
JP 8291199 A 19961105 JP 9697872 A 19960419 C07K-016/28 199703

Priority Applications (No Type Date): FR 954809 A 19950421
Filing Details:
Patent Kind Filing Notes Application Patent
EP 738736 A1
Designated States (Regional): AT CH DE FR GB IT LI NL SE
Language, Pages: EP 738736 (F, 16); FR 2733250 (24); JP 8291199 (9)

3/3/12 (Item 12 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

010947936
WPI Acc No: 96-444886/199645
XRAM Acc No: C96-140057
New recombinant rat obese gene - used to develop prods. to study obesity and to diagnose obesity and obesity factors
Patent Assignee: TAKEDA CHEM IND LTD (TAKE )
Inventor: FUJISAWA Y; NAKAO K; OGAWA Y
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
EP 736599 A2 19961009 EP 96105353 A 19960403 C12N-015/12 199645 B
EP 736599 A3 19961211 EP 96105353 A 19960403 C12N-015/12 199707
JP 8333394 A 19961217 JP 9679916 A 19960402 C07K-014/47 199709

Priority Applications (No Type Date): JP 9577966 A 19950403
Filing Details:
Patent Kind Filing Notes Application Patent
EP 736599 A2
Designated States (Regional): AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
Language, Pages: EP 736599 (E, 26); JP 8333394 (11)

3/3/13 (Item 13 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1997 Derwent Info Ltd. All rts. reserv.

010915789
WPI Acc No: 96-412740/199641
XRAM Acc No: C96-130090
XRPX Acc No: N96-347423
New immuno-receptor tyrosine-inhibitory motif peptide(s) - used to develop methods for regulating inflammatory and immune responses and for drug screening
Patent Assignee: NAT JEWISH CENT IMMUNOLOGY & RESPIRATORY (NAJE-N)
Inventor: CAMBIER J C
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9626961 A2 19960906 WO 96US2289 A 19960228 C07K-014/705 199641 B
AU 9652967 A 19960918 AU 9652967 A 19960228 C07K-014/705 199701
WO 9626961 A3 19970410 WO 96US2289 A 19960228 C07K-014/705 199729

Priority Applications (No Type Date): US 95397628 A 19950228
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9626961 A2
Designated States (National): AU CA JP US
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
AU 9652967 A Based on WO 9626961
Language, Pages: WO 9626961 (E, 99)

3/3/14 (Item 14 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010895727
WPI Acc No: 96-392678/199639
Related WPI Acc No: 92-366185; 93-036323; 93-182479; 93-405021; 95-005894; 97-235228
XRAM Acc No: C96-123565
Anti-foetal liver kinase 2 (flk-2) *antibodies* - useful in assays, for isolating *haematopoietic* stem cells expressing *receptor* and for obtaining ligands
Patent Assignee: UNIV PRINCETON (UYPR-N)
Inventor: LEMISCHKA I R
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| US 5548065 | A | 19960820 | US 91679666 | A | 19910402 | C07K-016/18 | 199639 B |
| | | | US 91728913 | A | 19910628 | | |
| | | | US 91793065 | A | 19911115 | | |
| | | | US 91813593 | A | 19911224 | | |
| | | | US 92906397 | A | 19920626 | | |
| | | | US 92975049 | A | 19921112 | | |
| | | | US 92977451 | A | 19921119 | | |
| | | | US 9355269 | A | 19930430 | | |
| | | | US 94252517 | A | 19941031 | | |

Priority Applications (No Type Date): US 92977451 A 19921119; US 91679666 A 19910402; US 91728913 A 19910628; US 91793065 A 19911115; US 91813593 A 19911224; US 92906397 A 19920626; US 92975049 A 19921112; US 9355269 A 19930430; US 94252517 A 19941031
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| US 5548065 | A | CIP of | US 91679666 | |
| | | CIP of | US 91728913 | |
| | | CIP of | US 91793065 | |
| | | CIP of | US 91813593 | |
| | | CIP of | US 92906397 | |
| | | CIP of | US 92975049 | |
| | | Div ex | US 92977451 | |
| | | Div ex | US 9355269 | |
| | | CIP of | | US 5185438 |
| | | Div ex | | US 5270458 |
| | | Div ex | | US 5367057 |

Language, Pages: US 5548065 (50)

3/3/15 (Item 15 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010824598
WPI Acc No: 96-321551/199632
XRAM Acc No: C96-102318
Neuropeptide Y receptor modified in third intracellular domain - useful to identify modulators of *receptor* activity for treating e.g. *obesity* , diabetes, hypertension, congestive heart failure, etc.
Patent Assignee: MERCK & CO INC (MERI )
Inventor: CASCIERI M A; MACNEIL D J; STRADER C D
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9614331 | A1 | 19960517 | WO 95US14377 | A | 19951106 | C07H-021/04 | 199632 B |

Priority Applications (No Type Date): US 94335017 A 19941107
Filing Details:

Patent Kind Filing Notes Application Patent
WO 9614331 A1
Designated States (National): CA JP US
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
Language, Pages: WO 9614331 (E, 59)

3/3/16 (Item 16 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010682946
WPI Acc No: 96-179901/199618
XRAM Acc No: C96-056786
Human *haematopoietin* *receptor* Hu-B1.219 - useful in design of molecular probes for prenatal testing and cancer diagnosis
Patent Assignee: PROGENITOR INC (PROG-N)
Inventor: CIOFFI J; SHAFER A W; SNODGRASS R H; ZUPANCIC T J; SNODGRASS H R
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9608510 | A1 | 19960321 | WO 95US10965 | A | 19950830 | | 199618 B |
| AU 9534194 | A | 19960329 | AU 9534194 | A | 19950830 | | 199628 |
| EP 730606 | A1 | 19960911 | EP 95931007 | A | 19950830 | | 199641 |
| | | | WO 95US10965 | A | 19950830 | | |
| EP 730606 | A4 | 19970312 | EP 95931007 | A | 19950000 | | 199729 |
| US 5643748 | A | 19970701 | US 94306231 | A | 19940914 | | 199732 |

Priority Applications (No Type Date): US 94355888 A 19941214; US 94306231 A 19940914
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9608510 A1
Designated States (National): AM AU BB BG BR BY CA CN CZ EE FI GE HU IS JP KG KP KR KZ LK LR LT LV MD MG MK MN MX NO NZ PL RO RU SG SI SK TJ TM TT UA UZ VN
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
AU 9534194 A Based on WO 9608510
EP 730606 A1 Based on WO 9608510
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
Language, Pages: WO 9608510 (E, 67); EP 730606 (E); US 5643748 (23)

3/3/17 (Item 17 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010197513 **Image available**
WPI Acc No: 95-098767/199513
XRAM Acc No: C95-044992
XRPX Acc No: N95-077933
New pure human erythropoietin receptor fragment - obtd. by expression as a fusion protein having a thrombin proteolytic cleavage site
Patent Assignee: LEE J Y (LEEJ-I)
Inventor: LEE J Y
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9505469 | A1 | 19950223 | WO 94US9298 | A | 19940815 | C12N-015/62 | 199513 B |
| JP 9501833 | W | 19970225 | WO 94US9298 | A | 19940815 | C12N-015/09 | 199718 |
| | | | JP 95507168 | A | 19940815 | | |
| EP 776370 | A1 | 19970604 | EP 94927925 | A | 19940815 | C12N-015/62 | 199727 |
| | | | WO 94US9298 | A | 19940815 | | |

Priority Applications (No Type Date): US 93106815 A 19930816

Filing Details:
Patent Kind Filing Notes Application Patent
WO 9505469 A1
Designated States (National): CA JP KR
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
JP 9501833 W Based on WO 9505469
EP 776370 A1 Based on WO 9505469
Designated States (Regional): AT BE DE FR GB IT NL SE
Language, Pages: WO 9505469 (E, 42); JP 9501833 (45); EP 776370 (E)

3/3/18 (Item 18 from file: 351)
DIALOG(R)File 351:DERWENT WPI


010180974 **Image available**
WPI Acc No: 95-082227/199511
XRAM Acc No: C95-036967
New haematopoietic stem cell lines with specific differentiation properties - made by transfected stem cells with nucleic acid encoding dominant negative suppressor of the retinoic acid *receptor* alpha, useful e.g. for *haematopoietic* reconstitution
Patent Assignee: HUTCHINSON CANCER RES CENT FRED (HUTC-N)
Inventor: COLLINS S J; TSAI S
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9504143 A1 19950209 WO 94US8450 A 19940728 C12N-015/12 199511 B
AU 9475152 A 19950228 AU 9475152 A 19940728 C12N-015/12 199521

Priority Applications (No Type Date): US 9399242 A 19930728
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9504143 A1
Designated States (National): AM AT AU BB BG BR BY CA CH CN CZ DE DK ES FI GB GE HU JP KE KG KP KR KZ LK LT LU LV MD MG MN MW NL NO NZ PL PT RO RU SD SE SI SK TJ TT UA US UZ VN
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE
AU 9475152 A Based on WO 9504143
Language, Pages: WO 9504143 (E, 100)

3/3/19 (Item 19 from file: 351)
DIALOG(R)File 351:DERWENT WPI


009488944
WPI Acc No: 93-182479/199322
Related WPI Acc No: 92-366185; 93-036323; 93-405021; 95-005894; 96-392678; 97-235228
XRAM Acc No: C93-080827
Totipotent *haematopoietic* stem cell *receptors*, their ligands and DNA sequences - for treating anaemia(s) and bone marrow damage due to e.g. cancer chemotherapy or radiotherapy
Patent Assignee: UNIV PRINCETON (UYPR-N)
Inventor: LEMISCHKA I R
Patent Family:
Patent No Kind Date Applicat No Kind Date Main IPC Week
WO 9310136 A1 19930527 WO 92US9893 A 19921116 C07H-015/12 199322 B
AU 9331394 A 19930615 AU 9331394 A 19921116 199340

Priority Applications (No Type Date): US 91793065 A 19911115
Filing Details:
Patent Kind Filing Notes Application Patent
WO 9310136 A1

Designated States (National): AU CA FI HU JP KP NO RO RU
Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT LU MC NL SE
AU 9331394 A Based on WO 9310136
Language, Pages: WO 9310136 (E, 127)

3/3/20 (Item 20 from file: 351)
DIALOG(R)File 351:DERWENT WPI


009072535
WPI Acc No: 92-199954/199224
XRAM Acc No: C92-090989
Regulation of haematopoietic cell association with skin - during chronic inflammation, using agents which target skin-associated leukocytes or inhibit CLAM-1 binding to cutaneous associated cells
Patent Assignee: UNIV LELAND STANFORD JUNIOR (STRD )
Inventor: BERG E; BUTCHER E C; PICKER L; BERG E L
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9208490 | A1 | 19920529 | WO 91US8512 | A | 19911114 | A61K-039/395 | 199224 B |
| EP 557424 | A1 | 19930901 | WO 91US8512 | A | 19911114 | A61K-039/395 | 199335 |
| | | | EP 92900490 | A | 19911114 | | |
| EP 557424 | A4 | 19940608 | EP 92900490 | A | 19920000 | A61K-039/395 | 199531 |

Priority Applications (No Type Date): US 90614616 A 19901116
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9208490 | A1 | | | |
| | | Designated States (National): CA JP | | |
| | | Designated States (Regional): AT BE CH DE DK ES FR GB GR IT LU NL SE | | |
| EP 557424 | A1 | Based on | WO 9208490 | |
| | | Designated States (Regional): CH DE FR GB LI | | |

Language, Pages: WO 9208490 (E, 39); EP 557424 (E)

3/3/21 (Item 21 from file: 351)
DIALOG(R)File 351:DERWENT WPI


009039792
WPI Acc No: 92-167154/199220
XRAM Acc No: C92-076890
Polypeptides similar to v-mlp protein of MPLV - for diagnosis and treatment of myeloproliferative diseases
Patent Assignee: INSERM INST NAT SANTE & RECH MEDICALE (INRM ); INSERM INST NAT SANTE & RECH MED (INRM )
Inventor: CHARON M; GISSELBRECHT S; PENCIOLELLI J F; SOUYRI M; TAMBOURIN P; VARLET P; VIGON I; WENDLING F
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9207074 | A1 | 19920430 | WO 90FR762 | A | 19901019 | C12N-015/12 | 199220 B |
| JP 6501915 | W | 19940303 | JP 90515169 | A | 19901019 | C07K-013/00 | 199414 |
| | | | WO 90FR762 | A | 19901019 | | |

Priority Applications (No Type Date): WO 90FR762 A 19901019
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9207074 | A1 | | | |
| | | Designated States (National): CA JP US | | |
| JP 6501915 | W | Based on | WO 9207074 | |

Language, Pages: WO 9207074 (F, 75); JP 6501915 (22)

3/3/22 (Item 22 from file: 351)

DIALOG(R)File 351:DERWENT WPI


008806357
WPI Acc No: 91-310369/199142
XRAM Acc No: C91-134422
Synergistic combination of two *monoclonal* *antibodies* - for blocking human transferrin receptor for inhibition of growth of proliferating cells e.g. leukaemia
Patent Assignee: SALK INST BIOLOGICAL STUDIES (SALK ); UNIV CALIFORNIA (REGC )
Inventor: TAETLE R; TROWBRIDGE I S; WHITE S N; WHITE S
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9114452 | A | 19911003 | | | | B | 199142 B |
| AU 9176670 | A | 19911021 | | | | B | 199203 |
| EP 525005 | A1 | 19930203 | EP 91907581 | A | 19910326 | B | 199305 |
| | | | WO 91US2031 | A | 19910326 | | |
| JP 5505823 | W | 19930826 | JP 91507366 | A | 19910326 | B | 199339 |
| | | | WO 91US2031 | A | 19910326 | | |
| AU 642014 | B | 19931007 | AU 9176670 | A | 19910326 | B | 199346 |
| EP 525005 | B1 | 19970611 | EP 91907581 | A | 19910326 | B | 199728 |
| | | | WO 91US2031 | A | 19910326 | | |
| DE 69126526 | E | 19970717 | DE 626526 | A | 19910326 | B | 199734 |
| | | | EP 91907581 | A | 19910326 | | |
| | | | WO 91US2031 | A | 19910326 | | |

Priority Applications (No Type Date): US 90500035 A 19900327
Filing Details:

| Patent | Kind | Filing Notes | Application | Patent |
|---|---|---|---|---|
| WO 9114452 | A | | | |
| | | Designated States (National): AU CA JP | | |
| | | Designated States (Regional): AT BE CH DE DK ES FR GB GR IT LU NL SE | | |
| EP 525005 | A1 | Based on | | WO 9114452 |
| | | Designated States (Regional): AT BE CH DE DK ES FR GB GR IT LI LU NL SE | | |
| JP 5505823 | W | Based on | | WO 9114452 |
| AU 642014 | B | Previous Publ. | | AU 9176670 |
| | | Based on | | WO 9114452 |
| EP 525005 | B1 | Based on | | WO 9114452 |
| | | Designated States (Regional): AT BE CH DE DK ES FR GB GR IT LI LU NL SE | | |
| DE 69126526 | E | Based on | | EP 525005 |
| | | Based on | | WO 9114452 |

Language, Pages: EP 525005 (E, 39); JP 5505823 (10); EP 525005 (E, 14)

3/3/23 (Item 23 from file: 351)
DIALOG(R)File 351:DERWENT WPI


008547338
WPI Acc No: 91-051401/199107
XRAM Acc No: C91-021861
XRPX Acc No: N91-039767
New hybrid mol. comprising erythropoietin receptor super-family cytokine - useful in imaging of cells or cell receptors
Patent Assignee: SERAGEN INC (SERA-N)
Inventor: SVRLUGA R C; WATES C A
Patent Family:

| Patent No | Kind | Date | Applicat No | Kind | Date | Main IPC | Week |
|---|---|---|---|---|---|---|---|
| WO 9101004 | A | 19910124 | | | | | 199107 B |
| EP 433442 | A | 19910626 | EP 90911931 | A | 19900706 | | 199126 |
| JP 4501808 | W | 19920402 | JP 90511281 | A | 19900706 | | 199220 |
| EP 433442 | A4 | 19920805 | EP 90911931 | A | 19900000 | | 199523 |

Priority Applications (No Type Date): US 89376656 A 19890706
Filing Details:

Patent Kind Filing Notes Application Patent
WO 9101004 A
Designated States (National): CA JP
Designated States (Regional): AT BE CH DE DK ES FR GB IT LU NL SE
EP 433442 A
Designated States (Regional): AT BE CH DE ES FR GB IT LI LU NL SE
JP 4501808 W Based on WO 9101004
Language, Pages: JP 4501808 (8)

3/3/24 (Item 1 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00731493
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Compositions and methods for stimulating megakaryocyte growth and differentiation
Zusammensetzungen und Verfahren zur Anregung des Wachstums und der Differenzierung von Megakaryozyten
Compositions et procede pour la stimulation de la croissance et la differentiation des megacaryocytes
PATENT ASSIGNEE:
AMGEN INC., (923233), Amgen Center, 1840 Dehavilland Drive, Thousand Oaks, CA 91320-1789, (US), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Bartley, Timothy D., 2431 McCrea Road, Thousand Oaks, CA 91362, (US)
Bogenberger, Jakob M., 2242 Barbara Drive, Camarillo, CA 93010, (US)
Bosselman, Robert A., 3301 Baccarat Street, Thousand Oaks, CA 91362, (US)
Hunt, Pamela, 2431 McCrea Road, Thousand Oaks, CA 91362, (US)
Samal, Babru B., 1136 Broadview Drive, Moorpark, CA 93021, (US)
Kinstler, Olaf B., P.O. Box 271, Newbury Park, CA 91320, (US)
LEGAL REPRESENTATIVE:
Brown, John David et al (28811), FORRESTER & BOEHMERT Franz-Joseph-Strasse 38, D-80801 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 690127 A1 960103 (Basic)
APPLICATION (CC, No, Date): EP 95104745 950330;
PRIORITY (CC, No, Date): US 221768 940331; US 252628 940531; US 321488 941012; US 347780 941130
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/19; C07K-014/52; C07K-017/08; A61K-038/19; A61K-047/48;
ABSTRACT WORD COUNT: 122

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPAB96 | 507 |
| SPEC A | (English) | EPAB96 | 24430 |
| Total word count - document A | | | 24937 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 24937 |

3/3/25 (Item 2 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00712874
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Compositions and methods for stimulating megakaryocyte growth and differentiation.
Zusammensetzungen und Verfahren zur Anregung des Wachstums und der Differenzierung von Megakaryozyten.

Compositions et procedes pour la stimulation de la croissance et la differenciation des megacaryocytes.
PATENT ASSIGNEE:
AMGEN INC., (923233), Amgen Center, 1840 Dehavilland Drive, Thousand Oaks, CA 91320-1789, (US), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Bartley, Timothy D., 2431 McCrea Road, Thousand Oaks, CA 91362, (US)
Bogenberger, Jakob M., 2242 Barbara Drive, Camarillo, CA 93010, (US)
Bosselman, Robert A., 3301 Baccarat Street, Thousand Oaks, CA 91362, (US)
Hunt, Pamela, 2431 McCrea Road, Thousand Oaks, CA 91362, (US)
Samal, Babru B., 1136 Broadview Drive, Moorpark, CA 93021, (US)
Kinstler, Olaf B., P.O.Box 271, Newbury Park, CA 91320, (US)
LEGAL REPRESENTATIVE:
Brown, John David et al (28811), FORRESTER & BOEHMERT Franz-Joseph-Strasse 38, D-80801 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 675201 A1 951004 (Basic)
APPLICATION (CC, No, Date): EP 95104711 950330;
PRIORITY (CC, No, Date): US 221768 940331; US 252628 940531; US 321488 941012; US 347780 941130
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/19; C07K-014/52; C12N-005/10; C07K-016/24; A61K-038/19; C07K-017/08; A61K-047/48;
ABSTRACT WORD COUNT: 108

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPAB95 | 1445 |
| SPEC A | (English) | EPAB95 | 24317 |
| Total word count - document A | | | 25762 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 25762 |

3/3/26 (Item 3 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00692880
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
*MONOCLONAL* *ANTIBODIES* THAT RECOGNIZE FLK-2 *RECEPTORS* AND THE ISOLATION OF PRIMITIVE *HEMATOPOIETIC* STEM CELL POPULATIONS
MONOKLONALE ANTIKORPER, DIE FLK-2-REZEPTOREN ERKENNEN, UND DIE ISOLIERUNG PRIMITIVER HAMATOPOIETISCHER STAMMZELLPOPULATIONEN
ANTICORPS MONOCLONAUX RECONNAISSANT LES RECEPTEURS FLK-2 ET ISOLEMENT DE POPULATIONS DE CELLULES SOUCHES PRIMITIVES HEMATOPOIETIQUES
PATENT ASSIGNEE:
IMCLONE SYSTEMS, INC., (1017470), 180 Varick Street, New York, NY 10014, (US), (applicant designated states: AT;BE;CH;DE;ES;FR;GB;IE;IT;LI;NL)
INVENTOR:
GOLDSTEIN, Neil I., 26 Kendal Avenue, Maplewood, NJ 07040, (US)
SONGSAKPHISAN, Ratchanee Apartment 6A, 67-47 Kissena Boulevard, Flushing, NY 11367, (US)
ROSE, Caroline Apartment 4F, 34-40 79 Street, Jackson Heights, NY 11372, (US)
LEGAL REPRESENTATIVE:
Grunecker, Kinkeldey, Stockmair & Schwanhausser Anwaltssozietat (100721), Maximilianstrasse 58, 80538 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 722489 A1 960724 (Basic)
WO 9507348 950316
APPLICATION (CC, No, Date): EP 94929168 940907; WO 94US10194 940907
PRIORITY (CC, No, Date): US 118468 930908
DESIGNATED STATES: AT; BE; CH; DE; ES; FR; GB; IE; IT; LI; NL
INTERNATIONAL PATENT CLASS: C12N-005/08; C12N-005/20; A61K-035/12;

A61K-035/14; A61K-035/28; A61K-039/395; G01N-033/53;
LANGUAGE (Publication,Procedural,Application): English; English; English

3/3/27 (Item 4 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00687588
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
NOVEL TYROSINE KINASE
NEUARTIGE TYROSINKINASE
NOUVELLE TYROSINE KINASE
PATENT ASSIGNEE:
Asahi Kasei Kogyo Kabushiki Kaisha, (219570), 2-6, Dojimahama 1-chome Kita-ku, Osaka-shi Osaka 530, (JP), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
SAKANO, Seiji, 351-1, Samejima, Fuji-shi, Shizuoka-ken 416, (JP)
LEGAL REPRESENTATIVE:
Boeters, Hans Dietrich, Dr. et al (2193), Patentanwalte Boeters & Bauer, Bereiteranger 15, 81541 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 732398 A1 960918 (Basic)
WO 9506113 950302
APPLICATION (CC, No, Date): EP 94925009 940825; WO 94JP1411 940825
PRIORITY (CC, No, Date): JP 93210403 930825; JP 9458553 940329
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-009/12; C12N-015/00;
ABSTRACT WORD COUNT: 133

LANGUAGE (Publication,Procedural,Application): English; English; Japanese
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPAB96 | 358 |
| SPEC A | (English) | EPAB96 | 10580 |
| Total word count - document A | | | 10938 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 10938 |

3/3/28 (Item 5 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00669066
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
*MONOCLONAL* *ANTIBODY*, PROCESS FOR PRODUCING THE SAME, AND USE THEREOF.
MONOKLONALE ANTIKORPER, ANWENDUNG UND VERFAHREN ZUR HERSTELLUNG.
ANTICORPS *MONOCLONAL*, PROCEDE DE PRODUCTION, ET UTILISATION.
PATENT ASSIGNEE:
TORAY INDUSTRIES, INC., (203533), 2-1, Nihonbashi-Muromachi 2-chome Chuo-ku, Tokyo 103, (JP), (applicant designated states: DE;FR;GB;IT)
INVENTOR:
NISHIKAWA, Satomi, 23-25-703, Takano-Higashihirakicho 1-chome, Sakyo-ku, Kyoto-shi, Kyoto 606, (JP)
SUDO, Tetsuo, Toray Industries Residence I-1, 3-6, Tsunishi 2-chome, Kamakura-shi, Kanagawa 248, (JP)
OKANO, Kiyoshi, 4-8-211, Ichinomiya 5-chome, Samukawa-cho, Kosa-gun, Kanagawa 253-01, (JP)
IZAWA, Akiko, 55-5-403, Katsura-cho, Sakae-ku, Yokohama-shi, Kanagawa 247, (JP)
NAKAMURA, Noriko, 5-1, Katase 5-chome, Fujisawa-shi, Kanagawa 251, (JP)
AKIYAMA, Naoko, 1101-15, Hon-machida, Machida-shi, Tokyo 194, (JP)
LEGAL REPRESENTATIVE:
Kador & Partner (100211), Corneliusstrasse 15, D-80469 Munchen, (DE)

PATENT (CC, No, Kind, Date): EP 667395 A1 950816 (Basic)
EP 667395 A1 960703
WO 9428160 941208
APPLICATION (CC, No, Date): EP 94917140 940601; WO 94JP887 940601
PRIORITY (CC, No, Date): JP 93130825 930601
DESIGNATED STATES: DE; FR; GB; IT
INTERNATIONAL PATENT CLASS: C12P-021/08; C07K-015/28; G01N-033/577; G01N-033/53; A01K-067/027; C12N-005/06; C12P-021/08; C12R-001/91
ABSTRACT WORD COUNT: 181

LANGUAGE (Publication,Procedural,Application): English; English; Japanese

3/3/29 (Item 6 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS
(c) 1997 EUROPEAN PATENT OFFICE. All rts. reserv.

00664616
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Receptors of interleukin-12 and *antibodies*.
Interleukin-12 Rezeptoren und Antikorper.
Recepteurs de l'interleukine 12 et anticorps.
PATENT ASSIGNEE:
F. HOFFMANN-LA ROCHE AG, (1107063), Grenzacherstrasse 124, CH-4002 Basel, (CH), (applicant designated states:
AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Chizzonite, Richard Anthony, 104 Richards Road, South Kent Connecticut 06785, (US)
Chua, Anne On, 5 Karen Court, Wayne New Jersey 07470, (US)
Gubler, Ulrich Andreas, 4 Inness Place, Glen Ridge New Jersey 07028, (US)
Truitt, Theresa Patricia, 109 Garner Avenue, Bloomfield New Jersey 07003, (US)
LEGAL REPRESENTATIVE:
Mezger, Wolfgang, Dr. et al (26171), Grenzacherstrasse 124 Postfach 3255, CH-4002 Basel, (CH)
PATENT (CC, No, Kind, Date): EP 638644 A1 950215 (Basic)
APPLICATION (CC, No, Date): EP 94110657 940708;
PRIORITY (CC, No, Date): US 94649 930719; US 94713 930719; US 248532 940531
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/12; C07K-014/715; C07K-016/28; A61K-038/17; A61K-039/395; G01N-033/68;
ABSTRACT WORD COUNT: 18

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 793 |
| SPEC A | (English) | EPABF2 | 15580 |
| Total word count - document A | | | 16373 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 16373 |

3/3/30 (Item 7 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS
(c) 1997 EUROPEAN PATENT OFFICE. All rts. reserv.

00651577
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Ligands for FLT3 receptors.
Ligante fur die FLT3 Rezeptoren.
Liants pour les recepteurs FLT3.
PATENT ASSIGNEE:
IMMUNEX CORPORATION, (485033), 51 University Street, Seattle Washington

98101, (US), (applicant designated states:
AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Lyman, Stewart D., 312 N. 4th Street, Seattle, Washington 98115, (US)
Beckmann, M. Patricia, 5454 Ragan Lane, Poulsbo, Washington 98370, (US)
LEGAL REPRESENTATIVE:
Roberts, Alison Christine et al (72641), Kilburn & Strode, 30 John Street , London WC1N 2DD, (GB)
PATENT (CC, No, Kind, Date): EP 627487 A2 941207 (Basic)
EP 627487 A3 960821
APPLICATION (CC, No, Date): EP 94303575 940519;
PRIORITY (CC, No, Date): US 68394 930524; US 106463 930812; US 111758 930825; US 162407 931203; US 209502 940307; US 243545 940511
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/12; C07K-013/00; C12P-021/08; A61K-037/02; C12N-015/87;
ABSTRACT WORD COUNT: 91

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 668 |
| SPEC A | (English) | EPABF2 | 15742 |
| Total word count - document A | | | 16410 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 16410 |

3/3/31 (Item 8 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00629390
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
G-CSF analog compositions and methods.
G-CSF Analoge und Verfahren zu ihrer Herstellung.
Analogues de G-CSF et methodes pour les obtenir.
PATENT ASSIGNEE:
AMGEN INC., (923233), Amgen Center, 1840 Dehavilland Drive, Thousand Oaks, CA 91320-1789, (US), (applicant designated states:
AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Osslund, Timothy, 475 Vista Montana, Camarillo, California 93010, (US)
LEGAL REPRESENTATIVE:
Vossius, Volker, Dr. et al (12524), Dr. Volker Vossius
Patentanwaltskanzlei - Rechtsanwaltskanzlei Holbeinstrasse 5, D-81679 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 612846 A1 940831 (Basic)
APPLICATION (CC, No, Date): EP 94101207 940127;
PRIORITY (CC, No, Date): US 10099 930128
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/27; C07K-003/00; C12P-021/02; C07K-013/00; G06F-015/60;
ABSTRACT WORD COUNT: 91

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 2177 |
| SPEC A | (English) | EPABF2 | 16120 |
| Total word count - document A | | | 18297 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 18297 |

3/3/32 (Item 9 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00619004
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
BISPECIFIC IMMUNOADHESINS
BISPEZIFISCHE IMMUNOADHESINE
IMMUNOADHESINES BISPECIFIQUES
PATENT ASSIGNEE:
GENENTECH, INC., (210480), 460 Point San Bruno Boulevard, South San Francisco California 94080, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IE;LI;LU;MC;SE)
INVENTOR:
ASHKENAZI, Avi, J., 1580 Tarrytown Street, San Mateo, CA 94402, (US)
CHAMOW, Steven, M., 300 Cupertino Way, San Mateo, CA 94403, (US)
LEGAL REPRESENTATIVE:
Greaves, Carol Pauline et al (50413), Mewburn Ellis York House 23 Kingsway, London WC2B 6HP, (GB)
PATENT (CC, No, Kind, Date): EP 656064 A1 950607 (Basic)
EP 656064 B1 970305
WO 9404690 940303
APPLICATION (CC, No, Date): EP 93920182 930817; WO 93US7783 930817
PRIORITY (CC, No, Date): US 931811 920817
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IE; LI; LU; MC; SE
INTERNATIONAL PATENT CLASS: C12N-015/62; C12N-015/13; C12N-015/12; C07K-019/00; C07K-016/28; C07K-016/10;
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS B | (English) | EPAB97 | 1142 |
| CLAIMS B | (German) | EPAB97 | 1200 |
| CLAIMS B | (French) | EPAB97 | 1326 |
| SPEC B | (English) | EPAB97 | 18728 |
| Total word count - document A | | | 0 |
| Total word count - document B | | | 22396 |
| Total word count - documents A + B | | | 22396 |

3/3/33 (Item 10 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00595202
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Peptide conjugate.
Peptide-Konjugate.
Conjugues peptidiques.
PATENT ASSIGNEE:
TAKEDA CHEMICAL INDUSTRIES, LTD., (204706), 1-1, Doshomachi 4-chome, Chuo-ku, Osaka 541, (JP), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;NL;PT;SE)
INVENTOR:
Fukuta, Makoto, 2-10-B-508, Tsurumainishi-machi, Nara, Nara 631, (JP)
Iinuma, Satoshi, 1-1-308, Dohshodai 1-chome, Suma-ku, Kobe, Hyogo 654, (JP)
Okada, Hiroaki, 44-11-704, Yamadaminami, Suita, Osaka 565, (JP)
LEGAL REPRESENTATIVE:
von Kreisler, Alek, Dipl.-Chem. et al (12437), Patentanwalte von Kreisler-Selting-Werner Bahnhofsvorplatz 1 (Deichmannhaus), D-50667 Koln, (DE)
PATENT (CC, No, Kind, Date): EP 599303 A2 940601 (Basic)
APPLICATION (CC, No, Date): EP 93118961 931125;
PRIORITY (CC, No, Date): JP 92318031 921127
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; NL;

PT; SE
INTERNATIONAL PATENT CLASS: A61K-047/48; A61K-043/00;
ABSTRACT WORD COUNT: 82

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 290 |
| SPEC A | (English) | EPABF2 | 5988 |
| Total word count - document A | | | 6278 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 6278 |

3/3/34 (Item 11 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00594796
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
ECK receptor ligands.
ECK-Rezeptor-Liganden.
Ligands de recepteurs ECK.
PATENT ASSIGNEE:
AMGEN INC., (923233), Amgen Center, 1840 Dehavilland Drive, Thousand Oaks, CA 91320-1789, (US), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Bartley, Timothy D., 2431 McCrea Road, Thousand Oaks, CA 91362, (US)
Fox, Gary M., 35 West Kelly Road, Newbury Park, CA 91320, (US)
Boyle, William J., 13024 Williams Ranch Road, Moorpark, CA 93021, (US)
Welcher, Andrew A., 707 Danvers Circle, Newbury Park, CA 91320, (US)
Parker, Vann P., 1086 Antelope Place, Newbury Park, CA 91320, (US)
LEGAL REPRESENTATIVE:
Vossius, Volker, Dr. et al (12524), Dr. Volker Vossius, Patentanwaltskanzlei-Rechtsanwaltskanzlei Holbeinstrasse 5, D-81679 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 597503 A2 940518 (Basic)
EP 597503 A3 950524
APPLICATION (CC, No, Date): EP 93118469 931115;
PRIORITY (CC, No, Date): US 977708 921113; US 145616 931109
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12N-015/12; C07K-013/00; G01N-033/68; G01N-021/55; A61K-037/02;
ABSTRACT WORD COUNT: 71

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 750 |
| SPEC A | (English) | EPABF2 | 11615 |
| Total word count - document A | | | 12365 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 12365 |

3/3/35 (Item 12 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00579011
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Intron/Exon structure of the human and mouse Beta 3 Adrenergic receptors genes.
Intron/Exon Struktur der Genen von den menschlichen und Maus Beta 3

adrenergischen Rezeptoren.
Structure de introns/exons de genes codants pour les recepteurs beta 3 adrenergiques souris et humain.
PATENT ASSIGNEE:
CENTRE NATIONAL DE LA RECHERCHE SCIENTIFIQUE, (428836), 15, quai Anatole France, F-75007 Paris, (FR), (applicant designated states: DE;FR;GB;IT)
INVENTOR:
Emorine, Laurent, 30, rue de Dom Remi, F-75013 Paris, (FR)
Strosberg, A. Donny, 66, rue de Javel, F-75015 Paris, (FR)
Nahmias-Kaminski, Clara, 109, Bd. Beaumarchais, F-75003 Paris, (FR)
LEGAL REPRESENTATIVE:
Desaix, Anne et al (62911), Ernest Gutmann - Yves Plasseraud S.A. 3, rue Chauveau-Lagarde, F-75008 Paris, (FR)
PATENT (CC, No, Kind, Date): EP 600136 A1 940608 (Basic)
APPLICATION (CC, No, Date): EP 92403248 921201;
PRIORITY (CC, No, Date): EP 92403248 921201
DESIGNATED STATES: DE; FR; GB; IT
INTERNATIONAL PATENT CLASS: C12N-015/12
ABSTRACT WORD COUNT: 52

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF2 | 631 |
| SPEC A | (English) | EPABF2 | 6272 |
| Total word count - document A | | | 6903 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 6903 |

3/3/36 (Item 13 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00513536
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
GM-CSF inhibiting *antibodies*.
GM-CSF-hemmende Antikorper.
Anticorps inhibiteurs de GM-CSF.
PATENT ASSIGNEE:
Bristol-Myers Squibb Company, (205414), 345 Park Avenue, New York, N.Y. 10154, (US), (applicant designated states:
AT;BE;CH;DE;DK;ES;FR;GB;GR;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
Bursuker, Isia, 29 Currier Way, Cheshire, CT 06410, (US)
Greenfield, Robert S., 10 Seiter Hill Road, Wallingford, CT 06492, (US)
Braslawsky, Gary R., 124 Heritage Drive, Glastonbury, CT 06033, (US)
LEGAL REPRESENTATIVE:
Kinzebach, Werner, Dr. et al (6468), Patentanwalte Reitstotter, Kinzebach und Partner Sternwartstrasse 4 Postfach 86 06 49, W-8000 Munchen 86, (DE)
PATENT (CC, No, Kind, Date): EP 499161 A2 920819 (Basic)
EP 499161 A3 930407
APPLICATION (CC, No, Date): EP 92102103 920207;
PRIORITY (CC, No, Date): US 653428 910211
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: C12P-021/08; A61K-039/395; C12N-005/20;
ABSTRACT WORD COUNT: 61

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF1 | 1062 |
| SPEC A | (English) | EPABF1 | 10214 |
| Total word count - document A | | | 11276 |

Total word count - document B 0
Total word count - documents A + B 11276

3/3/37 (Item 14 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00513145
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Improvements relating to growth factors.
Verbesserungen an Wachstumfaktoren.
Ameliorations relatives aux facteurs de croissance.
PATENT ASSIGNEE:
ICRF PATENTS LTD., (391401), Sardinia House, Sardinia Street, London WC2A 3NL, (GB), (applicant designated states: AT;BE;CH;DE;FR;GB;LI;LU;NL;SE)
YEDA RESEARCH AND DEVELOPMENT CO. LTD., (268946), P.O. Box 951, Rehovot 76100, (IL), (applicant designated states: AT;BE;CH;DE;FR;GB;LI;LU;NL;SE)
GENENTECH, INC., (210480), 460 Point San Bruno Boulevard, South San Francisco California 94080, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;LI;LU;NL;SE)
INVENTOR:
Waterfield, Michael D. c/o ICRF Patents LTD., Sardinia House, Sardinia Street, London WC2A 3NL, (GB)
Schlessinger, J. c/o Yeda Research and Dev.Co., P.O.Box 951, IL-Rehovot 76100, (IL)
Ullrich,Axel c/o Genentech, Inc., 460 Point San Bruno Boulevard, South San Francisco CA 94080, (US)
LEGAL REPRESENTATIVE:
Goldin, Douglas Michael et al (31061), J.A. KEMP & CO. 14, South Square Gray's Inn, London WC1R 5EU, (GB)
PATENT (CC, No, Kind, Date): EP 491675 A1 920624 (Basic)
APPLICATION (CC, No, Date): EP 92101700 850130;
PRIORITY (CC, No, Date): GB 8402379 840130; GB 8500538 850109
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: G01N-033/574; C12Q-001/68; C07K-015/00; C12P-021/00; C07K-007/04; A61K-049/00; A61K-039/395;
ABSTRACT WORD COUNT: 90

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF1 | 1009 |
| SPEC A | (English) | EPABF1 | 10965 |

Total word count - document A 11974
Total word count - document B 0
Total word count - documents A + B 11974

3/3/38 (Item 15 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00504372
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
METHODS FOR REMOVING LIGANDS FROM A PARTICLE SURFACE.
VERFAHREN ZUR ENTFERNUNG VON LIGANDEN VON EINER TEILCHEN-OBERFLACHE.
PROCEDES SERVANT A ENLEVER DES ANTICORPS D'UNE SURFACE DE PARTICULE.
PATENT ASSIGNEE:
CELLPRO INCORPORATED, (1437070), 22322-20th Avenue S.E.,, Bothell, WA 98021, (US), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IT;LI;LU;NL;SE)
INVENTOR:
BERENSON, Ronald, J., 6127 - 84th Avenue S.E., Mercer Island, WA 98040, (US)

PETERSON, Dale, R., 18630 - 28th Avenue, Bothell, WA 89012, (US)
LEGAL REPRESENTATIVE:
Armitage, Ian Michael et al (27761), MEWBURN ELLIS York House 23 Kingsway , London WC2B 6HP, (GB)
PATENT (CC, No, Kind, Date): EP 526577 A1 930210 (Basic)
EP 526577 B1 941130
WO 9116088 911031
APPLICATION (CC, No, Date): EP 91909347 910423; WO 91US2785 910423
PRIORITY (CC, No, Date): US 513056 900423
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: A61M-001/36;
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS B | (English) | EPBBF1 | 459 |
| CLAIMS B | (German) | EPBBF1 | 497 |
| CLAIMS B | (French) | EPBBF1 | 575 |
| SPEC B | (English) | EPBBF1 | 3776 |
| Total word count - document A | | | 0 |
| Total word count - document B | | | 5307 |
| Total word count - documents A + B | | | 5307 |

3/3/39 (Item 16 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00373006
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Lymphokines.
Lymphokine.
Lymphokines.
PATENT ASSIGNEE:
THE WISTAR INSTITUTE, (319700), 36th Street at Spruce, Philadelphia Pennsylvania 19104, (US), (applicant designated states: AT;BE;CH;DE;ES;FR;GB;GR;IT;LI;LU;NL;SE)
INVENTOR:
Defreitas, Elaine C., 731 Newton Road, Villanova, PA 19085, (US)
Abrams, J. Todd, 212 Dudley Avenue, Narberth, PA 19072, (US)
LEGAL REPRESENTATIVE:
Dean, John Paul et al (72771), Withers & Rogers 4 Dyer's Buildings Holborn, London EC1N 2JT, (GB)
PATENT (CC, No, Kind, Date): EP 392122 A1 901017 (Basic)
APPLICATION (CC, No, Date): EP 89313135 891215;
PRIORITY (CC, No, Date): US 284558 881215
DESIGNATED STATES: AT; BE; CH; DE; ES; FR; GB; GR; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: A61K-035/14; A61K-035/22; C12P-021/00;
ABSTRACT WORD COUNT: 51

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS A | (English) | EPABF1 | 686 |
| SPEC A | (English) | EPABF1 | 4347 |
| Total word count - document A | | | 5033 |
| Total word count - document B | | | 0 |
| Total word count - documents A + B | | | 5033 |

3/3/40 (Item 17 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00289799
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
Substituted imidazole derivatives and their preparation and use.

Substituierte Imidazol-Derivate und Verfahren zu deren Herstellung und deren Anwendung.
Derives d'imidazole substitue et leur preparation et utilisation.
PATENT ASSIGNEE:
ORION-YHTYMA OY, (252960), P.O. Box 425, SF-20101 Turku, (FI), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)
INVENTOR:
Karjalainen, Arto Johannes, Myllyojantie 13 H 37, SF-90650 Oulu 65, (FI)
Virtanen, Raimo Einari, Knaapintie 2-4, SF-21290 Rusko, (FI)
Karjalainen, Arja Leena, Tiilitie 15 C 24, SF-90650 Oulu 65, (FI)
Kurkela, Kauko Oiva Antero, Keulatie 4, SF-90560 Oulu 56, (FI)
LEGAL REPRESENTATIVE:
Collier, Jeremy Austin Grey et al (29481), J.A.Kemp & Co. 14, South Square Gray's Inn, London WC1R 5EU, (GB)
PATENT (CC, No, Kind, Date): EP 310745 A2 890412 (Basic)
EP 310745 A3 891011
EP 310745 B1 930127
APPLICATION (CC, No, Date): EP 88106210 851121;
PRIORITY (CC, No, Date): GB 8429578 841123
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: C07D-233/60; A61K-031/415; C07D-405/04;
ABSTRACT WORD COUNT: 44

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS B | (English) | EPBBF1 | 374 |
| CLAIMS B | (German) | EPBBF1 | 367 |
| CLAIMS B | (French) | EPBBF1 | 379 |
| SPEC B | (English) | EPBBF1 | 9352 |
| Total word count - document A | | | 0 |
| Total word count - document B | | | 10472 |
| Total word count - documents A + B | | | 10472 |

3/3/41 (Item 18 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00248079
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
4(5)-Substituted imidazole derivatives, and intermediates and processes for their preparation.
4(5)-Substituierte Imidazol-Derivate und Zwischenverbindungen und Verfahren zu ihrer Herstellung.
Derives de l'imidazole 4(5)-substitue ainsi que produits intermediaires et procedes pour leur preparation.
PATENT ASSIGNEE:
Orion-yhtyma Oy, (252968), Orionintie 1, PL 65, SF-02101 Espoo, (FI), (applicant designated states: AT;BE;CH;DE;ES;FR;GB;GR;IT;LI;LU;NL;SE)
INVENTOR:
Karjalainen, Arja Leena, Tiilitie 15 C 24, SF-90650 Oulu 65, (FI)
Karjalainen, Arto Johannes, Myllyojantie 13 H 37, SF-90650 Oulu 65, (FI)
LEGAL REPRESENTATIVE:
Sexton, Jane Helen et al (59301), J.A. Kemp & Co. 14 South Square Gray's Inn, London WC1R 5LX, (GB)
PATENT (CC, No, Kind, Date): EP 247764 A1 871202 (Basic)
EP 247764 B1 910814
APPLICATION (CC, No, Date): EP 87304304 870514;
PRIORITY (CC, No, Date): FI 862039 860515; FI 87462 870204
DESIGNATED STATES: AT; BE; CH; DE; ES; FR; GB; GR; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: C07D-233/56; C07D-233/64; C07C-069/753; C07C-061/39; C07C-061/40; C07C-059/86; C07C-049/563; A61K-031/415; C07C-255/47;
ABSTRACT WORD COUNT: 136

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS B | (English) | EPBBF1 | 2404 |
| CLAIMS B | (German) | EPBBF1 | 2333 |
| CLAIMS B | (French) | EPBBF1 | 2767 |
| SPEC B | (English) | EPBBF1 | 4798 |
| Total word count - document A | | | 0 |
| Total word count - document B | | | 12302 |
| Total word count - documents A + B | | | 12302 |

3/3/42 (Item 19 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00190782
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
IMPROVEMENTS RELATING TO GROWTH FACTORS.
VERBESSERUNGEN AN WACHSTUMSFAKTOREN.
AMELIORATIONS RELATIVES AUX FACTEURS DE CROISSANCE.
PATENT ASSIGNEE:
IMPERIAL CANCER RESEARCH TECHNOLOGY LIMITED, (391402), Sardinia House, Sardinia Street, London WC2A 3NL, (GB), (applicant designated states: AT;BE;CH;DE;FR;GB;LI;LU;NL;SE)
INVENTOR:
WATERFIELD, Michael, D. Imperial Cancer, Research Fund P.O. Box 123 Lincoln's Inn Fields, London WC2A 3PX, (GB)
SCHLESSINGER, J., The Weizmann Institute of Science, Rehovot, (IL)
ULLRICH, Axel, Genentech Inc. 460 Point San Bruno Boulevard, South San Francisco, CA 94080, (US)
LEGAL REPRESENTATIVE:
Cresswell, Thomas Anthony et al (50352), J.A. Kemp & Co. 14 South Square Gray's Inn, London WC1R 5EU, (GB)
PATENT (CC, No, Kind, Date): EP 171407 A1 860219 (Basic)
EP 171407 B1 931118
WO 8503357 850801
APPLICATION (CC, No, Date): EP 85900716 850130; WO 85GB45 850130
PRIORITY (CC, No, Date): GB 8402379 840130; GB 8500538 850109
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: G01N-033/574; C12Q-001/68; C07K-015/00; C12P-021/00; C07K-007/04; A61K-049/00; A61K-039/395;
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

| Available Text | Language | Update | Word Count |
|---|---|---|---|
| CLAIMS B | (English) | EPBBF1 | 287 |
| CLAIMS B | (German) | EPBBF1 | 257 |
| CLAIMS B | (French) | EPBBF1 | 343 |
| SPEC B | (English) | EPBBF1 | 9011 |
| Total word count - document A | | | 0 |
| Total word count - document B | | | 9898 |
| Total word count - documents A + B | | | 9898 |

3/3/43 (Item 20 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS


00080010
**ORDER fax of complete patent from KR SourceOne. See HELP ORDER348**
*Monoclonal* *antibodies* specific for the human transferrin receptor glycoprotein.
Fur das menschliche Transferrinrezeptor-Glykoprotein spezifische monoklonale Antikorper.
Anticorps monoclinaux specifiques pour la glycoproteine receptrice de la transferinne humaine.

PATENT ASSIGNEE:
THE SALK INSTITUTE FOR BIOLOGICAL STUDIES, (273850), 10010 North Torrey Pines Road, La Jolla California 92038, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;NL;SE)
INVENTOR:
Trowbridge, Ian, 3105 Duccomun Avenue, San Diego California, (US)
LEGAL REPRESENTATIVE:
Lawrence, Malcolm Graham et al , Malcolm Lawrence & Co. 9th Floor Terminus House Terminus Street, Harlow Essex CM20 1XF, (GB)
PATENT (CC, No, Kind, Date): EP 79696 A1 830525 (Basic)
EP 79696 B1 880309
APPLICATION (CC, No, Date): EP 82305658 821025;
PRIORITY (CC, No, Date): US 315194 811026
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; NL; SE
INTERNATIONAL PATENT CLASS: C12P-001/00; C12N-015/00; C12N-005/00; A61K-039/395; C12R-001/91
ABSTRACT WORD COUNT: 142

LANGUAGE (Publication,Procedural,Application): English; English; English

3/3/44 (Item 1 from file: 377)
DIALOG(R)File 377:Derwent Drug File


00469740 DERWENT ACCESSION NUMBER: 92-10247
In Vivo Interleukin-1 (IL-1) Administration Indirectly Promotes Type II IL-1 *Receptor* Expression on *Hematopoietic* Bone Marrow Cells: Novel Mechanism for the Hematopoietic Effects of IL-1.
Dubois C M; Ruscetti F W; Keller J R; Oppenheim J J; Hestdal K; Chizzonite R
Roche DynCorp
Blood 78, No. 11, 2841-47, 1991

3/3/45 (Item 2 from file: 377)
DIALOG(R)File 377:Derwent Drug File


00438702 DERWENT ACCESSION NUMBER: 91-30915
Molecular Mechanisms of Insulin Resistance.
Pillay T S; Makgoba M W
S.Afr.Med.J. 79, No. 10, 607-13, 1991